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SEPTEMBER

THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION

Relations of Human Denture—*Orton & Lischer*
Dental Infection and Osteomyelitis—*Wilensky*
Mineral Metabolism—*Moore*
Pressure and Force—*Richmond*
Gold Foil Study Club—*Hollenback*
Impression Taking and Materials—*Wright*
Periodontal Diseases—*Willman*
Mouth in Pregnancy—*Reed*
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1933 Meeting
The Convention: An Interpretation
National Board of Dental Examiners
COUNCIL ON DENTAL THERAPEUTICS
Accepted Dental Remedies

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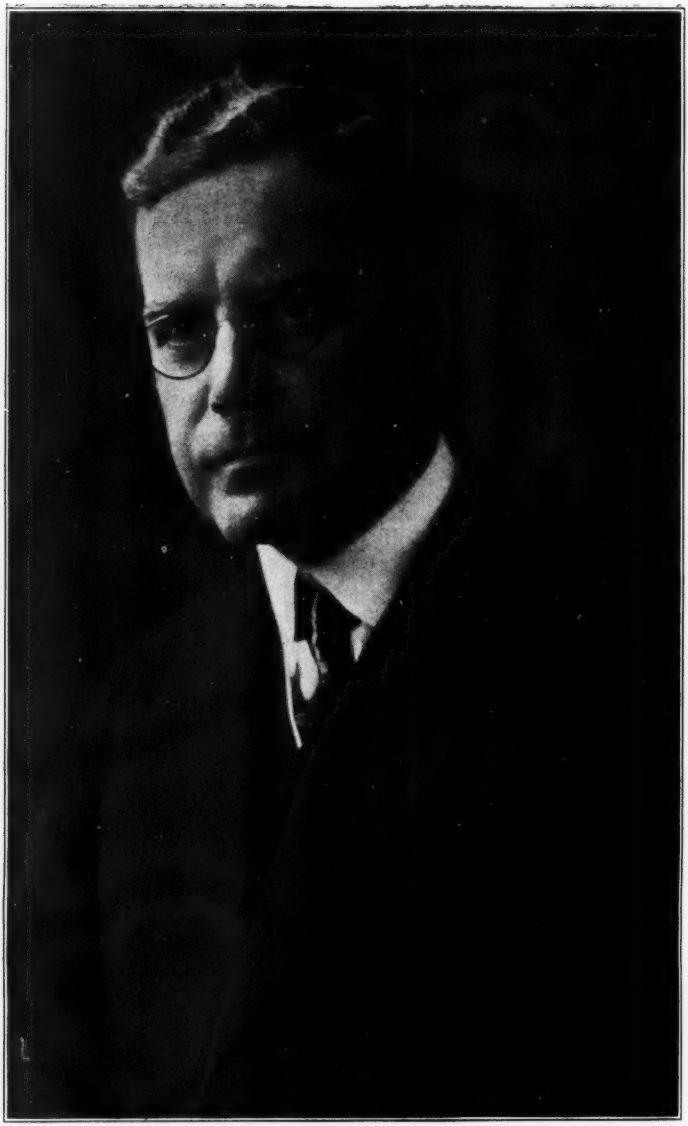

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President Elect of the American Dental Association

THE JOURNAL
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Original Communications

RELATIONS OF THE HUMAN DENTURE*

By FORREST H. ORTON,† D.D.S., and B. E. LISCHER, D.M.D., San Francisco, Calif.

THE basis for diagnosis of dental anomalies and of dental mutilations has undoubtedly been a concept of a "normal denture," i. e., of the most favorable relationship of the teeth of man. While the dental profession has formulated various concepts of them, these dental relationships have never been firmly established. Indeed, most of our efforts in this respect have been characterized by "the too human desire to be *certain*, rather than to *know*."

In crown and bridge prosthesis, we provide mechanical treatment for mutilated dental arches to restore their function. Occasionally, some of our so-called "border-line" cases require dental orthopedic preparation. The orthodontic problem, on the other hand, is largely one of growth and development of the denture, and may even be extended into the field of prevention. Hence, the problems with which we are dealing would seem to have nothing in common. Nevertheless, these two divisions of dentistry do have the same interest in, and are equally concerned with, a scientific method of diagnosis.

Diagnostic classifications employed in crown and bridge prosthesis are largely based on the treatment employed, and consist of a substitution of a Latin nomenclature for English equivalents. In orthodontics, considerable progress has been achieved, though there exists widespread hesitancy in adopting the recently proposed cephalometric procedures of Simon.

We selected the problems of determining norms of the most favorable relationships of the teeth as the base line from which to evaluate the numerous, less favorable deviations of the teeth and to verify, or add some new facts to, the concepts which had previously been presented. In other words, our aim was to add our bit to odontologic knowledge, to

*Read before the Porcelain Crown and Bridge Section at the Third Triennial Session of the Pacific Coast Dental Conference, Seattle, Wash., July 8, 1932.

†Deceased.

throw additional light, if possible, on the morphologic characteristics of the human denture, and on its relations to the human face.

Aside from any qualifications which we may or may not possess for pursuing this research, we at least comprehend our one advantage, namely, the fact that the abundant material is accessible to teachers in a large university, giving them an opportunity to collect data which are seldom, if ever, obtainable in private practice. Thus, in addition to teaching the available facts regarding a subject, it is the privilege of university teachers to add to this knowledge.

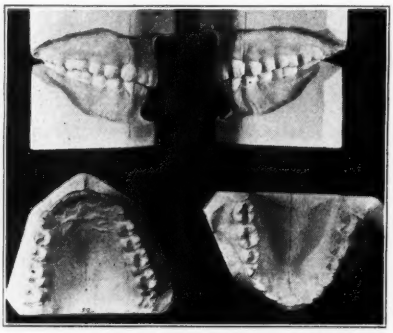


Fig. 1.—Plaster denture reproduction of Case 50. (To reduce the size of the illustrations, the photographs of the maxillary casts have been cut off. In the actual specimens, their bases represent the eye-ear planes of the individuals' heads.)

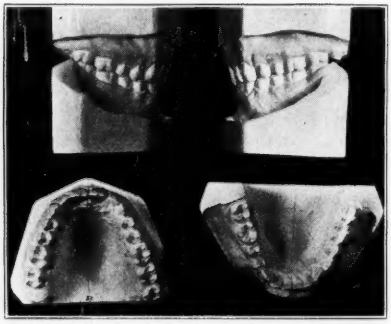


Fig. 2.—Plaster denture reproductions of Case 51.

In August, 1930, 2,982 students entered the university at Berkeley. In compliance with the rule that all students registering for the first time must submit to a thorough medical examination, the dental staff of the Cowell Memorial Hospital, where the examinations were conducted, found that 132 members of the class presented anatomically correct, unmutilated dentures. A separate list of this group was compiled by the clerk in charge and a letter was then addressed to each student requesting a second, more comprehensive dental examination (by the authors). That the latter examination was not obligatory, and its time requirements could easily conflict with the demands of class schedules, may account for the fact that only fifty-two students responded.

The conditions under which the examinations were conducted were ideal, the dental infirmary of the hospital having four dental operating rooms, with a complete equipment in each room, and a laboratory adequately equipped for our needs; the making of plaster reproductions of the teeth, mounting, transferring, etc.

Whole-hearted support and assistance was received on every hand. Mention must be made of the invaluable assistance given us by the director of the hospital, Dr. R. T. Legge; Dr. Ruby Cunningham; Drs. Margaret Black, of Berkeley, and Herbert R. Foster, of Oakland, for taking impressions: Drs. Allen H. Suggett and William S. Smith, of San Francisco, for the drawing of graphs, and Dr. Frederick W. Schubert, of San Francisco for preparation of the illustrations.

METHODS OF INVESTIGATION

Special appointments were arranged with the fifty-two students who responded, so that a more thorough investigation could be carried out. This consisted of the following:

Measurements were taken to determine the amount of overbite and overjet of all students examined (2,982). The method employed for measuring the overbite consisted simply in drawing a line on the labial surfaces of the mandibular incisors, guided by the cutting edges of the maxillary incisors, with the teeth in centric occlusion, and then measuring the distance from this line to the incisal margins of the mandibular incisors.

The method of measuring the overjet consisted simply of measuring the distance from the lingual surface of the incisal margin of the maxillary incisors to the labial surfaces of the mandibular incisors.

The search for examples of ideal, anatomically correct occlusion, was first conducted by direct observation, to eliminate all individuals presenting more or less malocclusion, and to compile the list for our more comprehensive examinations. All students whose dentures appeared to have a favorable mesiodistal relation of the buccal cusps when the teeth were in centric occlusion were placed on our list of 132 cases.

As our methods of investigation were conducted separately from this point, it

TABLE 1.—PERCENTAGE DISTRIBUTION OF AMOUNT OF OVERJET AND OVERBITE MEASURED IN MILLIMETERS IN 1,498 MEN ENTERING THE UNIVERSITY OF CALIFORNIA IN 1930

Overjet			Overbite		
Millimeters	Number	Percentage of Total	Millimeters	Number	Percentage of Total
—6	1	0.1	—6	1	0.1
—5	..	....	—5	2	0.1
—4	1	0.1	—4	4	0.3
—3	1	0.1	—3	6	0.4
—2	7	0.5	—2	5	0.3
—1	3	0.2	—1	6	0.4
0	210	14.0	0	139	9.3
1	443	29.6	1	252	16.8
2	439	29.3	2	330	22.0
3	212	14.2	3	395	26.3
4	63	4.2	4	165	11.0
5	41	2.7	5	110	7.4
6	20	1.3	6	33	2.2
7	14	0.9	7	30	2.0
8	10	0.7	8	10	0.6
9	4	0.3	9	6	0.4
10	24	1.6	10	3	0.2
11	1	0.1	11	1	0.1
12	5	0.2	12	..	....
13	..	....	13	..	....
14	1	0.1	14	..	....
Total	1,498	100.2	Total	1,498	99.9

was decided to take two separate sets of impressions. Since we believed it important to determine the relation of the protrusive, left and right centric occlusion, the usual custom of procuring so-called "study models" was followed (by F. H. O.) and they were oriented on instruments based on the spheroidal theory of occlusion. The three-point contact in protrusive, left and right centric occlusion was also recorded.

The orbitalia and left gonion were then marked (by B. E. L.) for photostatic facial reproductions, taken one-fourth natural size with a Simon photostatic camera. Thereafter, the mesiodistal diameters of the maxillary incisors were measured with sharp-pointed dividers for subsequent comparison of their dental arches with Pont's index of arch width. Gnathostatic impressions were then taken and complete gnathostatic denture reproductions were constructed of the fifty-two students. (Figs. 1 and 2.) Three-dimensional graphs were then drawn for each of these reproductions (Figs. 3-4) and the photostatic left profiles were all marked in accordance with Simon's recommendations.

TABLE 2. — PERCENTAGE DISTRIBUTION OF AMOUNT OF OVERJET AND OVERBITE MEASURED IN MILLIMETERS IN 1,587 WOMEN ENTERING THE UNIVERSITY OF CALIFORNIA IN 1930

Overjet			Overbite		
Millimeters	Number	Percentage of Total	Millimeters	Number	Percentage of Total
—4	..	....	—4	2	0.1
—3	..	....	—3	1	0.1
—2	2	0.1	—2	2	0.1
—1	5	0.3	—1	4	0.2
0	139	8.8	0	82	5.2
1	399	25.2	1	213	13.4
2	488	30.7	2	443	28.0
3	288	18.2	3	446	28.1
4	111	7.0	4	202	12.7
5	71	4.5	5	126	7.9
6	30	1.9	6	25	1.6
7	13	0.8	7	30	1.8
8	16	0.9	8	6	0.4
9	5	0.3	9	3	0.2
10	15	0.9	10	2	0.1
11	1	0.1	11	..	....
12	3	0.2	12	.	....
13	..	....	13	..	....
14	1	0.1	14	..	....
Total	1,587	100.0	Total	1,587	99.9

FINDINGS

1. *Photostatic.*—A. Proportions of the profile contour: (*a*) position of the cheilion (Fig. 5): 36 profiles revealed a mildly anterior position of the cheilion in relation to the orbital plane; 11 profiles recorded the cheilion on the orbital plane; 4 profiles showed the cheilion slightly posterior to the plane.

(*b*) position of the gnathion (Fig. 6): 13 profiles revealed a mildly anterior position of the gnathion; 27 profiles recorded the gnathion on the orbital plane; 11 profiles showed the gnathion posterior to this plane.

(*c*) behavior of the oral fissure in its relation to the horizontal plane (Fig. 7): 39 profiles revealed parallelism between the fissure and this plane; 11 profiles recorded a divergence posteriorly with the plane; 1 profile showed a convergence to posterior with the plane.

(*d*) upper lip curve (Figs. 8-9): 37 profiles revealed a gradual anterior curve (sn-ls) of the upper lip; 1 profile presented an abrupt curve of the upper lip; 8 profiles showed an absence of this curve; 5 profiles presented a receding upper lip (opisthocheilia), the angle prn-sn-ls being markedly obtuse.

(*e*) lower lip curve (Figs. 10-11): 37

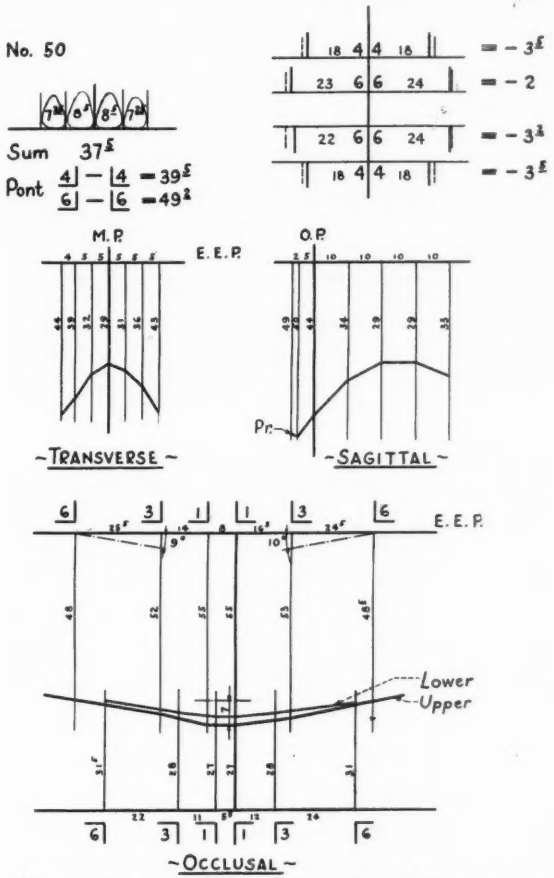


Fig. 3.—Graphs made from plaster reproductions of Case 50.

profiles presented a gradual curve of the lower lip; 12 profiles revealed an abrupt curve of this lip; 1 profile showed the absence of a curve in this lip; 1 profile exhibited a markedly receding lower lip.

B. Dimensions of the profile diagram:

(*b*) Tragion angle, or-t-go (Fig. 13): 88 degrees represents the largest angle found; 66 degrees, the smallest angle found; 75.9 degrees, the average of the series.

(*c*) Linear dimensions. (*a*) orbitale

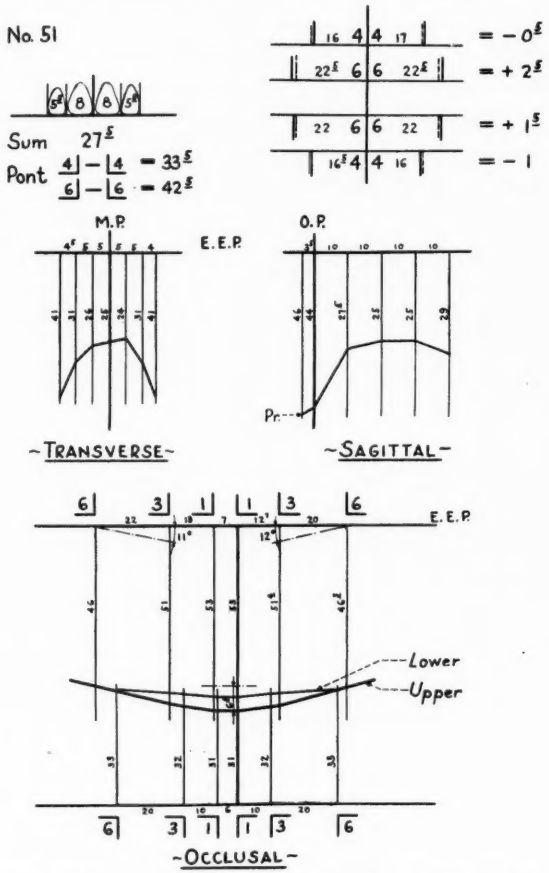


Fig. 4.—Graphs made from plaster reproductions of Case 51.

1. Angles. (*a*) gonion angle, t-go-gn (Fig. 12): 138 degrees represents the largest angle found; 112 degrees, the smallest angle found; 124 degrees, the average of the series.

to tragion, or-t (Fig. 14): 9.6 cm., longest measurement found; 7.2 cm., shortest measurement found; 8.3 cm., average of the series.

(*b*) orbitale to gnathion, or-gn (Fig.

15): 10 cm., longest measurement found; 7.6 cm., shortest measurement found; 8.5 cm., average of the series.

(*c*) tragion to gonion, t-go (Fig. 16): 8.4 cm., longest measurement found; 4.4 cm., shortest measurement found; 6.5 cm., average of the series.

(*d*) gonion to gnathion, go-gn (Fig. 17): 8 cm., longest measurement found; 6 cm., shortest measurement found; 7.1 cm., average of the series.

monized accurately; 21 dentures were slightly wider; 23 dentures were narrower (less) than the index measurements; 1 denture was less in the premolar region and wider in the molar region.

(*c*) Direction of the roots of the upper buccal teeth; 40 dentures revealed parallelism; 11 dentures disclosed a convergence of the apices toward the plane.

(*d*) palatal curve[1]: 4.1 cm., represents the widest curve; 2.8 cm., the narrowest

Fig. 5.—Variations of relation of cheilion to orbital plane.

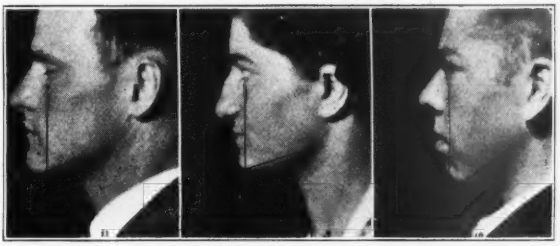


Fig. 6.—Variations of relation of gnathion to orbital plane.

2. *Gnathostatic.*—A. Relations of the dentures to the raphemedian plane: (*a*) Symmetry of the lateral halves: 20 dentures (0.392 per cent) presented accurate symmetrical relations; 15 dentures (0.294 per cent) presented slight deviations of 1 or 2 mm. constituting a group of "physiologic symmetry"; 16 dentures (0.313 per cent) revealed marked asymmetrical relations.

(*b*) Confirmation of measure points with the index of Pont: 6 dentures har-

curve; 3.47 cm., the average width of the series; 2 cm., the greatest median height of these curves; 1.1 cm., represents the least median height; 1.52 cm., the average median height of the series. (Figs. 18-19.)

1. A transverse curve, parallel to the orbital plane and 2 cm. posterior to it, was marked on each maxillary plaster reproduction with the aid of a symmetrograph. The transverse curves thus procured and transferred to their respective graphs were measured for width and height.

B. Relations of the dentures to the orbital plane[2]: (*a*) Symmetry: 41 dentures (0.803 per cent) revealed a striking symmetry in this respect, the orbital plane passing through identical points on the teeth of each side; 10 dentures (0.196 per cent) were asymmetrical.

(*b*) Prognathism[3]: 15 dentures (0.294 per cent) revealed an accurate confirmation; 16 dentures (0.313 per cent) revealed a mild degree of orthognathism, i.e., less than 5 mm.; 5 dentures (0.098 per cent) revealed a medium prognathism; 14 dentures (0.274 per cent) presented the prosthion posterior to the plane; i. e., were orthognathic; 2 dentures showed the prosthion on the orbital plane; 1 denture showed an anterior position of 15 mm.; 5.59 mm. represents the average for the series. (Fig. 20.)

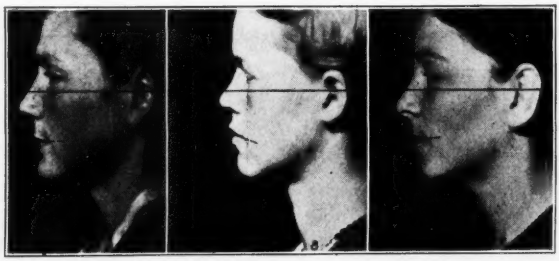


Fig. 7.—Variations of relation of oral fissure to horizontal eye-ear plane of head.

Fig. 8.—Variations of profile of upper lip.

(*c*) Direction of the maxillary incisor roots: 43 dentures revealed a striking parallelism between these roots and the orbital plane (Fig. 1); 6 dentures presented a posterior inclination of these roots; 2 dentures exhibited roots with an anterior inclination. (Fig. 2).

C. Relations of the dentures to the horizontal plane: (*a*) Median height of

2. An orbital plane, marked on the casts at right angles to the median plane, intersects the right and left sides of the dental arches and thus permits an evaluation of the relative sagittal symmetry of each lateral half.

3. The degree of prognathism of a given denture has long been a topic of compelling interest to orthodontists, but the craniometric methods of investigation employed by anthropometry were inapplicable to investigations on the living. Therefore Simon's norm was utilized for this phase of the examinations, i. e., it was assumed that the prosthion (pr) should lie 5 mm. anterior to the orbital (transverse) plane.

the upper occlusal curve: 9 mm. represents the greatest median height found; 1.5 mm., the smallest median height found; 5.5 mm., the average median height of the series. (Fig. 21.)

(*b*) Inclination of the molar cuspid secants of the maxillary occlusal curve: 18 degrees represents the greatest degree of inclination found; 2 degrees, the least amount of inclination found; 9.3 degrees, the average for the right secants of the series; 9.1 degrees, the average for the left secants of the series. (Fig. 22.)

(*c*) Form of the maxillary occlusal curve: 9 dentures presented curves characterized as deep; 27, curves of medium depth; 15, curves which were shallow.

(*d*) Direction of the roots: 43 dentures presented a perpendicular relation of the long axes of their roots; 6, a posterior inclination of the roots; 2, an anterior inclination.

(*e*) Length of ordinates from the horizontal plane: Prosthion ordinate, greatest length, 5.6 cm., shortest 4.0 cm.; orbital ordinate, greatest length, 5.6 cm., shortest, 3.5 cm.; 2 cm. posteriorly, greatest length, 3.5 cm., shortest, 2.2 cm.; 4 cm. posteriorly, greatest length, 3.8 cm., shortest, 2.2 cm. (Fig. 23.)

Fig. 9.—Variations of height of upper lip.

Fig. 11.—Variations of lower lip.

Fig. 10.—Variations of lower lip curve.

Fig. 12.—Case 10, presenting a large gonion angle, and Case 18, presenting a small gonion angle.

3. *Dynamic Relations of the Teeth.*—The spheroidal theory of occlusion has proved to be a very useful fiction in clinical practice. For this reason, we should take advantage of every opportunity to find out more about it. The theory has been summed up in the following definition:

In a well developed mandible (with all the teeth *in situ*), the incisal margins of the anterior teeth, and the points of the cusps of the posterior teeth, will conform approximately to the periphery of an 8-inch sphere. Such an ideal arrangement will, in all probability, never be found in nature. Some slight deviations are bound to occur. Only when these deviations are sufficient to interfere with the favorable relationship of the teeth to such an extent that the functions of the teeth are perverted are the deviations recognized as unfavorable, a condition hereafter known as "perverted axial stress."

Fig. 13.—Variations of tragion angle.

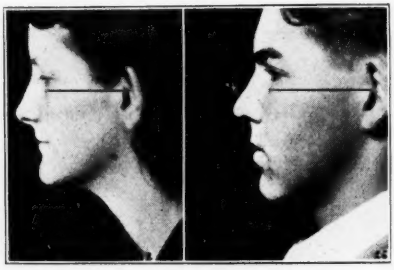


Fig. 14.—Extreme differences in length of line: orbital to tragion.

These findings deal principally with the dynamic relationship of the teeth, i. e., the empty gliding movements of the mandible when the lower denture is gliding against the upper denture.

The plaster reproductions of fifty-one students disclosed what is generally regarded as "normal occlusion." This concept is usually obtained by direct observation of the mesiodistal relation of the buccal cusps, the only part of the teeth within the range of vision when the teeth are in contact.

On further examination, student 52 was found to present a unilateral distoclusion and was therefore excluded from the investigation. The other records were all mounted on a Monson instrument. The measurements were transferred by means of a facebow. All were mounted to a 4-inch radius and checked by means of Needle's checkbite, until we were quite sure that the teeth in the upper and lower jaws bore the same cuspal relation on the instrument that they did in the mouth during the same movements of the mandible. Under such favorable conditions, we might expect to find additional verification of the spheroidal theory, also to determine to what extent a favorable relation of the teeth in centric occlusion could be used as a guide in determining a favorable relation of the teeth during the functional range of occlusion.

As might be expected, no cast was found to be ideal; that is to say, in none were all the teeth in the lower jaw in ideal cuspal contact in all relations with all the teeth in the opposing jaw, namely, the mesiodistal relationship of the buccal cusps, the mesiodistal relationship of the lingual cusps, and the buccolingual or transverse relationship of both the buccal and lingual cusps.

It is not an easy matter to so describe these three different relations of the teeth by the written word as to afford a total, coordinated image to the mind's eye. Perhaps this is one of the reasons that the

buccal relationship of the teeth is usually regarded as a sufficient guide to "normal occlusion."

Of the fifty-one mounted casts (all representing so-called "normal occlusion"), there were only eleven in which the lower denture could be so moved as to maintain an approximate occlusal contact with the opposing teeth during the functional gliding movements of the mandible. But, of the eleven casts, there were none with ideal cuspal contact in all relations; i.e., there were none in which all the buccal and lingual cusps on both sides of the arch were in ideal contact with their opposing teeth during the gliding movements of the lower denture. The lingual cusps of the upper teeth were the ones usually out of contact. Because of a fairly constant deviation in the direction of the long axis of the upper teeth from the direction of the long axis of the lower teeth, the spheroidal contact arrangement of the cusps of the upper teeth was changed, the buccal cusps becoming the longer and the lingual cusps the shorter, the buccolingual curve of occlusion being thus reversed at that point in the arch. None of these eleven approximately favorable dentures had an overbite of more than 1.5 mm. Five had an end-to-end bite.

Fig. 15.—Variations of facial height.

Notwithstanding the slight defects described, there was no interference with the empty gliding movements of the lower denture from centric occlusion throughout all the functional ranges of occlusion back to centric occlusion. It is our opinion that these eleven dentures approach as nearly to the ideal as can be expected under conditions of modern civilization.

Fig. 16.—Extreme differences in length of line: tragion to gonion.

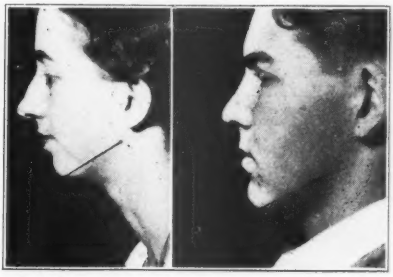


Fig. 17.—Variations of length in line: gonion to gnathion.

Of the remaining forty casts, none had what is known as a three-point contact during the prehensile, or protrusive, movements of the mandible.

Perverted axial stress was present to a greater or less degree in all the casts, either one side, both sides, or the cuspids and incisors. A condition of perverted axial stress was a frequent condition as regards the cuspids. Eight of the forty

cases presented a condition of perverted axial stress on all the teeth in the upper dentures.

continuous gliding movement of the lower denture, forcing the jaws apart, just as one long cog would interfere in the

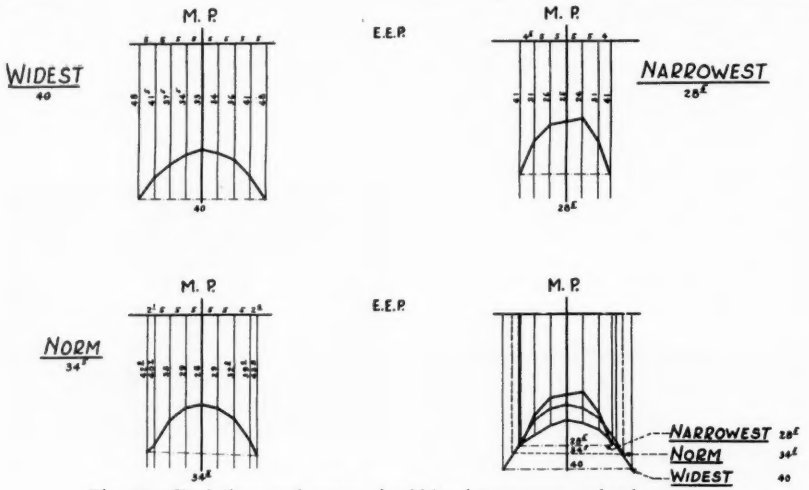


Fig. 18.—Variations and norm of width of transverse palatal curve.

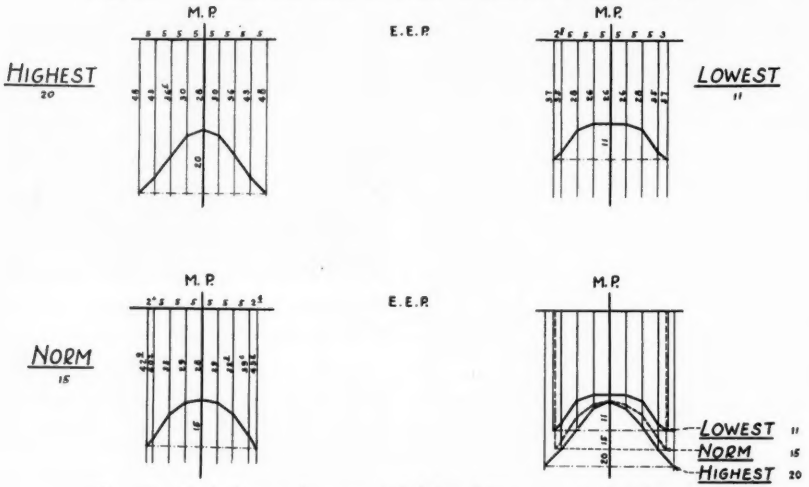


Fig. 19.—Variations and norm of height of transverse palatal curve.

All of the forty casts had an overbite of over 2 mm. None of these casts had sufficient overjet to compensate for 2 mm. overbite. The overbite interferes with smooth running mechanism of two cogged wheels forcing the wheels apart where the long cog came to the point of interdigitation.

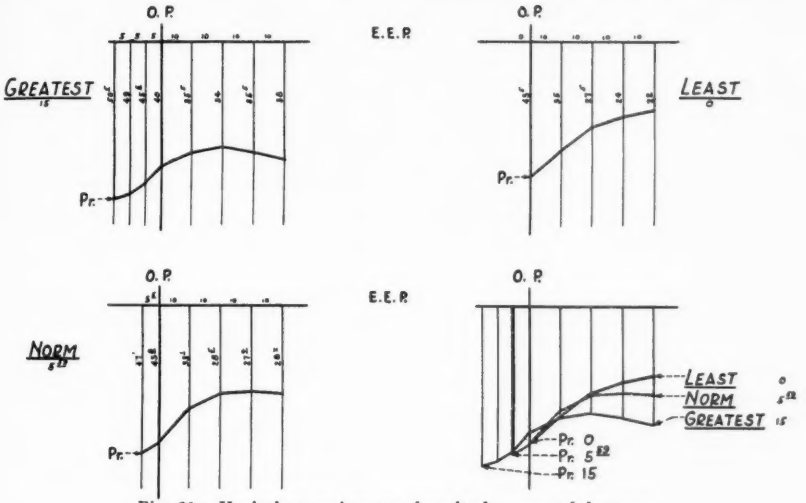


Fig. 20.—Variations and norm of sagittal curves of dentures.

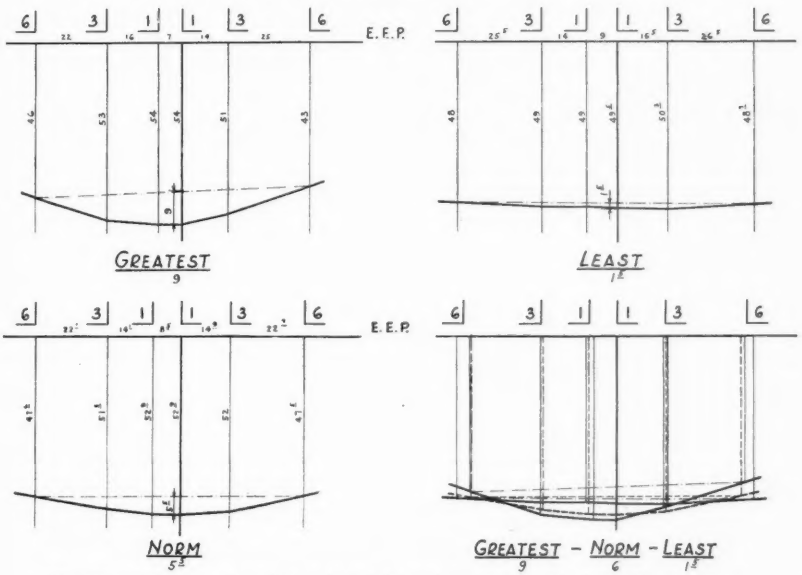


Fig. 21.—Extremes of variation in height and norm of occlusal maxillary curves.

Balanced occlusion was, of course, impossible during the gliding movements of the lower denture. Only during the static relation of the upper and lower denture (so-called centric occlusion) were the posterior teeth (distad from the cuspids)

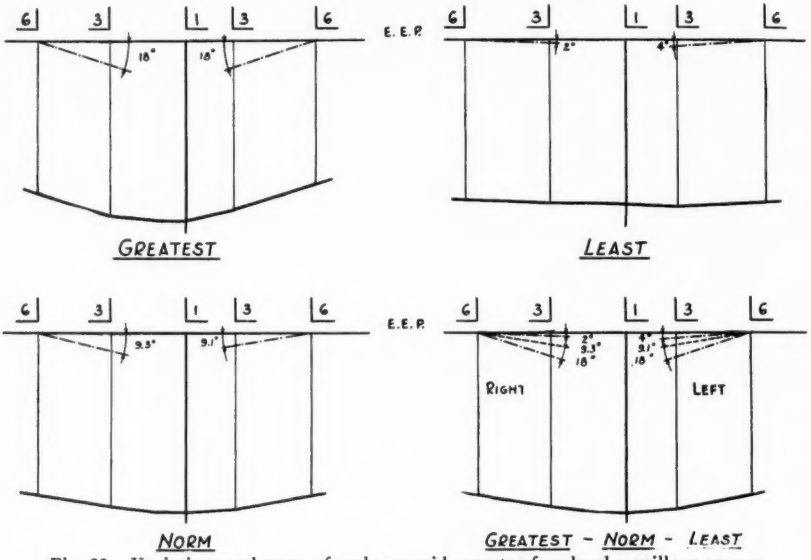


Fig. 22.—Variations and norm of molar-cuspid secants of occlusal maxillary curves.

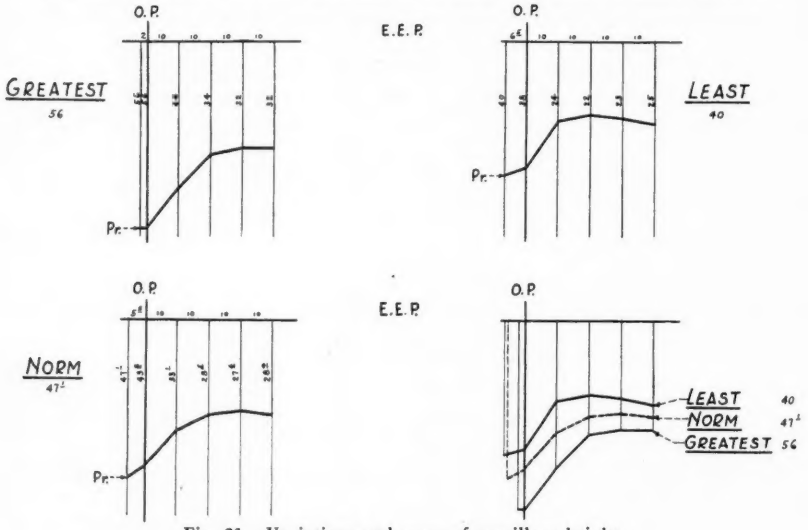


Fig. 23.—Variations and norm of maxillary height.

in contact, except occasionally on the buccal cusps.

CONCLUSIONS

As an organ which, primarily serves his physical needs, the denture of man is influenced not only by those conditions of life which make for health, growth and development, but also by those which

cause disease and deformity. A notable characteristic of dental and orthodontic generalization concerning the denture of man is the tacit assumption that it reveals an absolute, infallible ideal of perfection, as if it were in truth "the realization of some exterior, cosmic purpose." We believe that a larger acquaintance with biologic science and an unbiased observation of the variations which man's denture so constantly presents oblige us to discard the idea of any great natural fixed patterns, which the profession has so long regarded as "the culmination of a gradual evolutionary process." The fact that of 2,982 young men and women in pursuit of knowledge and culture, and whose previous nurture was not devoid of dental care, less than 5 per cent presented teeth which are usually called "normal," and that of this small number not one supplied a masticating organ which might be accepted as an archtype for that idealization that we fostered so fondly these many years, compels us to acknowledge our "ignorance of ultimate causes and designs."

We have observed that while occlusion is a characteristic of man's teeth "essentially dependent on anatomic conditions, and exhibits only slight variations," these mild and generally symmetrical deviations occur unceasingly. As Bateson so aptly stated in 1894, "Variability of a form is, so far as can be seen, as much a part of its specific characters as any other feature of its organization." Since there are no fixed points in the face and denture, and obviously there never will be any, the relationship of the one to the other can never be otherwise expressed than as average relationships derived from statistical summaries of individual measurements.

Our investigation also shows that "the normal does not exist in nature for man's judgment, but is created by him." To continue, in the words of Simon, we believe that "the most fundamental principle which reason can apply to the conception of the normal, and which it will permanently apply, is usefulness;" that "by its aid we are, in many instances, able to understand the real of the material world, to find our way and render practical treatments successful," and that "the true character, or nature of the norm-

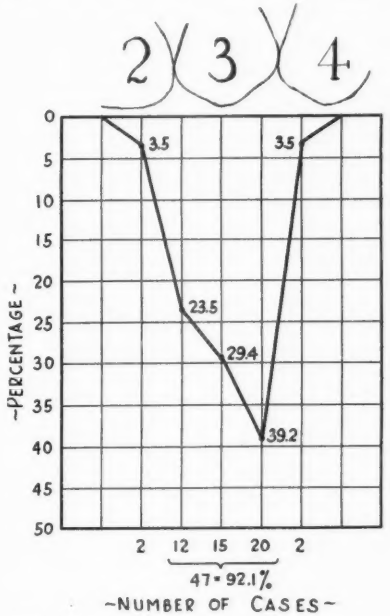


Fig. 24.—Norm patterns obtained from fifty-one cases.

concept is best understood if we conceive it as a fiction."

This places our problem in what John Morley calls "the narrow land of rational certainty: relative, conditional and experimental." But, in this limited region, theories need not be unchangeable and exempt from amendment or revision to be useful in advancing knowledge.

The foregoing survey of the human

denture was undertaken with the hope of throwing some additional light on the spheroidal theory of occlusion, by the aid of statistical methods to determine, if possible, how closely those individuals with anatomically correct dentures, i.e., where all the teeth in both dentures were in favorable relationship with each other in all relations in centric occlusion, would if we could determine accurately by this theory the most favorable relationships of the teeth, it should enable us to determine with equal accuracy those less favorable relationships usually found in clinical practice.

As has been stated, no ideal dentures were found, but those few which approached the ideal (eleven dentures)

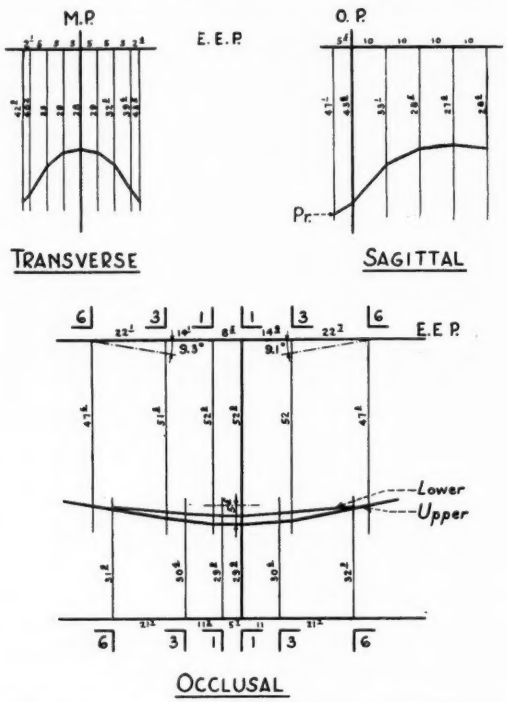


Fig. 25.—Frequency curve of relations of transverse, perpendicular orbital plane to fifty-one dentures.

conform to the spheroidal theory. Our first step was to transfer these fifty-one so-called normal casts to an instrument built to conform to the spheroidal theory of occlusion. Then, by applying the spheroidal measurements, we hoped to evaluate the practicability of this theory as an aid in diagnosis. We reasoned that also conformed more closely to the spheroidal theory than those which deviated more from the ideal. The most constant deviation from the ideal was an unfavorable overbite on one or more of the anterior teeth. The result of the overbite is to throw the stress exerted during the forward gliding movement of the den-

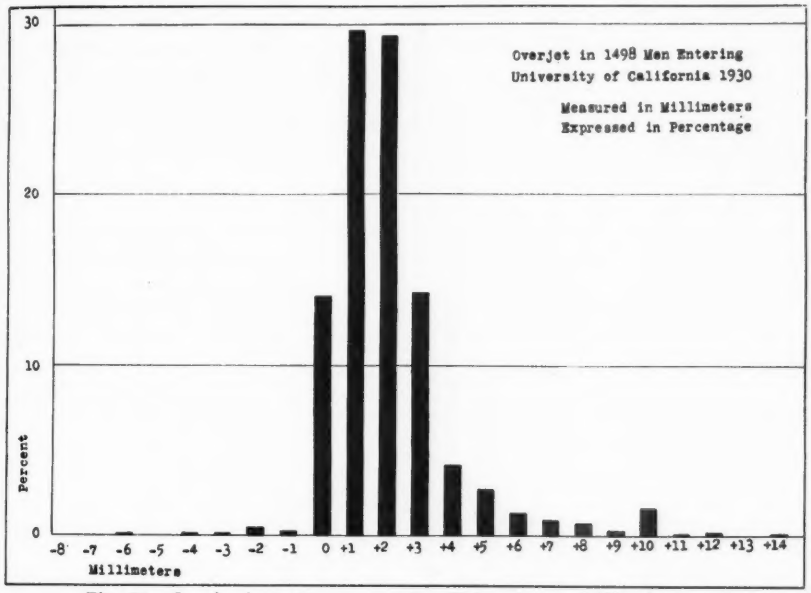
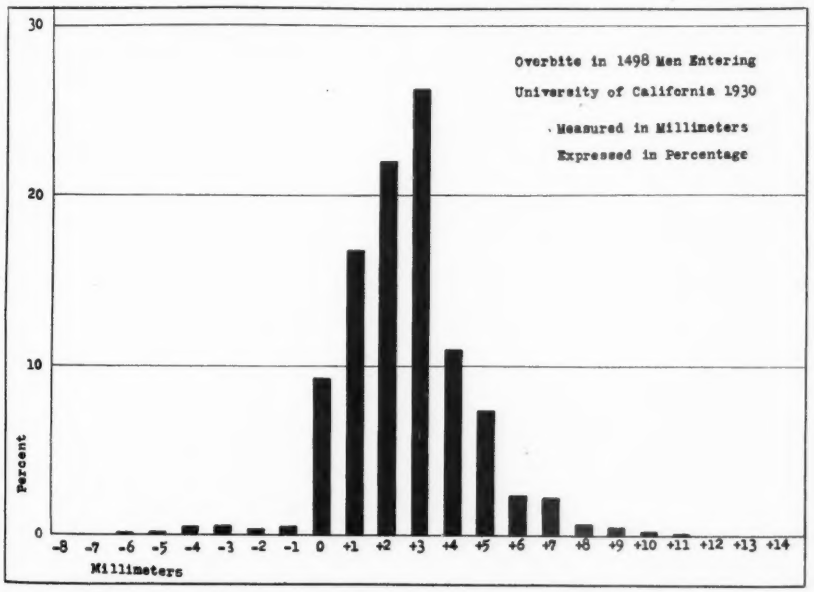

Overjet in 1498 Men Entering
University of California 1930
Measured in Millimeters
Expressed in Percentage
Percent
30
20
10
0
-8 -7 -6 -5 -4 -3 -2 -1 0 +1 +2 +3 +4 +5 +6 +7 +8 +9 +10 +11 +12 +13 +14
Millimeters

Fig. 26.—Overjet in 1,498 men entering University of California in 1930.


Overbite in 1498 Men Entering
University of California 1930
Measured in Millimeters
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30
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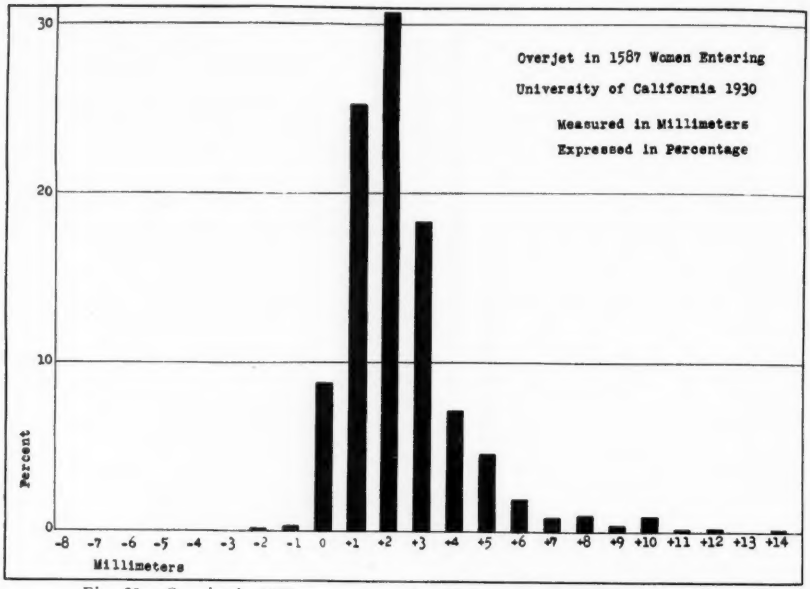
-8 -7 -6 -5 -4 -3 -2 -1 0 +1 +2 +3 +4 +5 +6 +7 +8 +9 +10 +11 +12 +13 +14
Millimeters

Fig. 27.—Overbite in 1,498 men entering University of California in 1930.


Overjet in 1587 Women Entering
University of California 1930
Measured in Millimeters
Expressed in Percentage
Percent
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20
10
0
-8 -7 -6 -5 -4 -3 -2 -1 0 +1 +2 +3 +4 +5 +6 +7 +8 +9 +10 +11 +12 +13 +14
Millimeters

Fig. 28.—Overjet in 1587 women entering University of California in 1930.

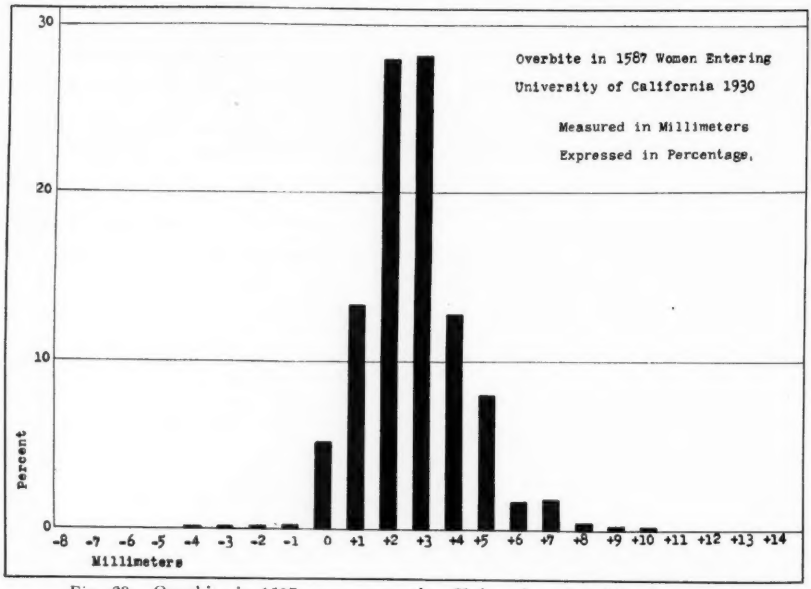
Overbite in 1587 Women Entering
University of California 1930
Measured in Millimeters
Expressed in Percentage.
Percent
30
20
10
0
-8 -7 -6 -5 -4 -3 -2 -1 0 +1 +2 +3 +4 +5 +6 +7 +8 +9 +10 +11 +12 +13 +14
Millimeters

Fig. 29.—Overbite in 1587 women entering University of California in 1930.

ture entirely on those few teeth in contact. Lacking any exact data, we cannot even estimate the amount of additional stress exerted on these teeth. A roentgenographic examination might have afforded some useful information in these excessive overbite cases.

Even the generous budget allowed by the university was not sufficient to permit the taking of roentgenographic films of all of these dentures. A careful examination by the direct method disclosed no objective symptoms of periodontal disease.

What effect the lack of cuspal contact on the posterior teeth due to excessive overbite has on masticating efficiency, i.e., whether it limits the masticating function of the posterior teeth to punching a bolus of food, we are unable to say because of our lack of exact data as to just how we chew. One fact we may conclude with certainty; namely, there can be no continuous contact of all the teeth in the denture during the gliding movements of the lower teeth, in those dentures in which the overbite is in excess of 2 mm., with no compensating overjet.

The gliding movement of the lower denture anteriorly (or, as we commonly say, forward) is prevented from continuing in this direction by the overbite. The jaw is forced downward at the same time, and thus the posterior teeth are forced apart.

Among many other things, this survey has forced on our convictions the fact of the utter inadequacy of diagnosing occlusal relationships by direct observation of the teeth. Even the so-called study model method, while permitting us to view the teeth in all relations, and in many cases to judge the amount of their deviation from the normal, does not permit us to judge whether the resultant deformities are due entirely to the loss of teeth, lack of development in the jaws, or both, or to what extent either of these factors may have contributed to the deformity. The superiority of the gnathostatic method as a diagnostic guide in evaluating these problems has been clearly demonstrated.

It is our opinion, if the present empiric art of partial denture construction is to progress to anything bordering on the scientific, it must concern itself with the collection of more scientific data on the subject of odontology and a more intensive training in this fundamental subject for the undergraduate student.

Our method of tabulating the overbite and overjet statistics collected during this survey is shown in the accompanying tables and Figures 26-29.

The amount of overbite and overjet presented is a statistical ratio of the entire group of students (2,982) taken by direct measurement, as heretofore described.

THE PATHOGENIC RELATIONSHIP BETWEEN DENTAL INFECTION AND OSTEOMYELITIS OF THE JAWS AND THE PRINCIPLES OF TREATMENT GOVERNING SUCH COMBINED LESIONS*

By ABRAHAM O. WILENSKY, M.D., New York City

THE close relationship which, in the vast majority of the cases, exists between various odontogenous conditions and osteomyelitis of the jaws, results directly from the pathogenic rôle which caries, infection and certain traumatic conditions of the teeth play in the production of acute infection of the jaw bones proper. In order to understand the production of the bone infection and to apply prophylactic measures, it is necessary to have full knowledge of the preceding odontogenous lesions from which the osteomyelitis results. Incidentally, I shall emphasize the marked similarity of the teeth and the bones proper as far as their structure, pathogenesis and inflammatory processes are concerned.

The anatomic structure of a typical tooth as given in Gray's Anatomy is as follows:

A longitudinal section of a tooth will show the presence of a central cavity corresponding in a general way to the crown of the tooth. Processes of the main chamber pass from its body, one down into each root, each one opening at the apex by a minute orifice, the apical foramen. This cavity is known as the pulp-chamber; the minute canals, as the pulp-canals. The cavity contains a soft, vascular and sensitive organ called the dental pulp. It is made up of myxomatous tissue, and contains numerous blood-vessels and nerves, which enter by way of the apical foramina. It does not possess lymphatics. The periphery of the pulp is bounded by a layer of cells arranged like columnar epithelium, each cell sending one or more branched processes through the basic substance of the dentine. These are the dentine-forming cells, the odontoblasts of Waldeyer. The blood-vessels break up into innumerable capillary loops which lie beneath the layer of odontoblasts. The nerve-fibrils break up into numberless non-medullary filaments, which spread out beneath the odontoblasts, and they probably send terminal filaments to the extreme periphery of the pulp outside of the odontoblasts.

The matrix cells and their processes are irregularly arranged in the body of the pulp, but in the canal portion the fibrillae are in the direction of the axis of the root.

Section of the tooth will exhibit three hard tissues in a tooth: one forming the greater mass of the tooth; hence its name dentine (the ivory); a layer which covers the exposed part of the dentine in the crown of the tooth, the enamel; and a thin layer, which is disposed on the surface of the fang, the cement or crusta petrosa; both cementum and enamel are thinnest at the neck and thickest upon their distal portions.

The ivory, or dentine, forms the principal mass of a tooth; in its central part is the cavity enclosing the pulp. It is a modification of osseous tissue, from which it differs, however, in structure. On microscopic examination it is seen to consist of a number of minute wavy and branching tubes having distinct parietes. They are called the dentinal tubuli; and are imbedded in a dense hematogenous substance, the intertubular tissue.

The enamel is the hardest and most compact part on a tooth, and forms a thin crust over the

*A lecture delivered to the Dental Staff of Mount Sinai Hospital, New York City, Nov. 15, 1932.

exposed part of the crown as far as the commencement of the root. It is thickest on the grinding surface of the crown until worn away by attrition, and becomes thinner toward the neck. It consists of minute hexagonal rods, columns, or prisms. They lie parallel with one another, resting by one extremity upon the dentine, which presents a number of minute depressions for their reception, and forming the free surface of the crown by the other extremity. Numerous minute interstices intervene between the enamel-fibers near their dentinal surface. It is noted in rare cases that the dentinal fibers penetrate a certain distance between the rods of the enamel. No nutritive canals exist in the enamel.

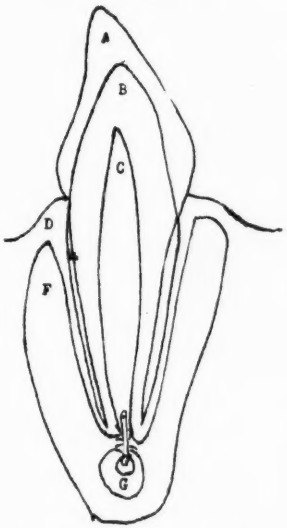

Fig. 1.—Structure of a typical tooth: *A*, enamel; *B*, dentin; *C*, pulp and pulp cavity; *D*, dental periosteum; *E*, cementum; *F*, bone; *G*, canal in bone carrying nutrient artery; *G*, nutrient artery with apical branch.

The cortical substance or cementum (crusta petrosa) is disposed as a thin layer on the roots of the teeth from the termination of the enamel as far as the apex of the root, where it is usually very thick. In structure and chemical composition it resembles bone. It contains, sparingly, the lacunae and canaliculi which characterize true bone; the lacunae placed near the surface have the canaliculi radiating from the side of the lacunae toward the peridental membrane (dental periosteum), and those more deeply placed join with adjacent dentinal tubuli. In the thicker portions of the crusta petrosa the lamellae and Haversian canals peculiar to bone are also occasionally found.

There is a remarkable general similarity between the gross structure of a tooth and that of an ordinary long bone. A long bone consists of a central cavity, the medullary cavity (which corresponds to the pulp cavity of a tooth), surrounded by layers of cancellous and cortical bone

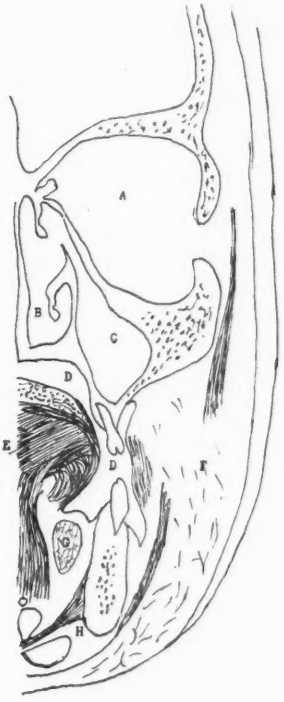

Fig. 2.—Cross-section of face and skull, showing anatomic relationships in spread of suppuration. *A*, orbit; *B*, nasal fossa; *C*, antrum of Highmore; *D*, mouth; *E*, substance of tongue; *F*, substance of cheek; *G*, one of salivary glands; *H*, submaxillary space.

(which correspond to the dentin and enamel of the tooth), all enclosed in a strong membrane, the periosteum (which corresponds to the peridental membrane). The medullary canal contains the marrow (corresponding to the pulp of the teeth) and marrow and pulp each contains blood vessels and nerves. If one took two teeth and joined them at their apices, one would have a typical long bone.

The similarity continues to a remarkable degree with the infections which occur in both the teeth and the bones; for instance, odontogenous conditions fall into the following groups.

SUPERFICIAL CARIES OF THE TEETH

Caries of the teeth begins in the nooks and crannies furnished by the irregularities of the crowns (i. e., the exposed portions) of the teeth. Particles of food gather there; ever present organisms—molds, fungi and streptococci of various strains—find a haven for growth; the

Fig. 3.

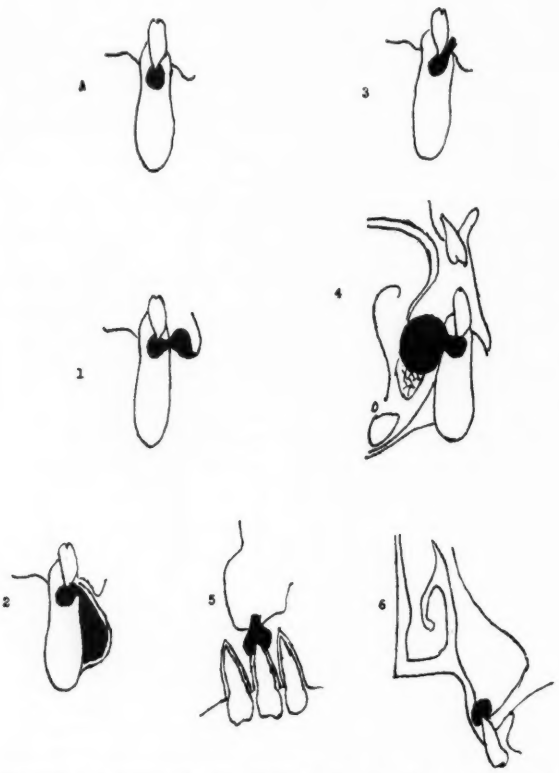

Fig. 3.—Directions of spread of dento-alveolar suppuration. *A*, periapical abscess.

superficial enamel and the underlying dentin soften and gradually become eroded by attrition; a superficial cavity results. Pain is present only when the nerve endings are exposed. As long as the pulp cavity is not entered, the integrity of the tooth is not diminished. No involvement of the jaw bone proper ever occurs with this variety of dental condition.

PULP CAVITY AND ROOT CANAL INVOLVEMENT

The pathology of the dental pulp has been most completely studied by Wedl, Slater and Black, and nearly all of the knowledge now possessed on this subject has come to us as a result of their researches. The available knowledge indicates that there are two distinctive varieties. Bacterial infection is often a source of acute hyperemia of the pulp. This is commonly caused by the invasion of the pulp chamber by the disintegrating processes of caries, originating as described in the immediately preceding section. It not infrequently occurs in those cases in which the pulp has not been exposed, but is still covered with a layer of decalcified and softened dentin (pseudo-exposure), the tubuli of which are filled with saprophytic and pyogenic organisms, which readily penetrate to the pulp and establish irritation, inflammation, suppuration and devitalization. A thin layer of sound dentin is a positive external protection against infection of the pulp with the pyogenic organisms.

When secondary involvement of the capillary vascular loops in the pulp cavity takes place, a much more heightened process results, during the course of which the tooth structure disintegrates much more quickly. Periapical abscess formation frequently forms a part of the picture. Usually, a cystic cavity results, involving more than one apical area.

PYORRHEA ALVEOLARIS

Pyorrhea alveolaris seems to be an atypical condition. A wide difference of opinion exists in reference to the etiology of this disease. Marshall has found, after a careful review of the literature, that the opinions expressed can all be grouped under four heads: 1. The disease originates in some constitutional state or dyscrasia. 2. It is caused entirely by local irritation and environmental conditions. 3. It is due to infection of the tissues with micro-organisms. 4. It is induced by deficient exercise of the teeth, gums and alevolar process.

Pyorrhea alveolaris primarily affects the pericementum, manifesting itself in a suppurative inflammation, sometimes acute in form, but generally of a chronic type. Secondarily, the inflammatory process involves the walls of the alveolus and the gum. The gum retracts from the side of the tooth; the potential space between root and tooth socket becomes an actual interval as the infective and irritative process spreads downward into the socket; coincidentally, the tooth loosens in the socket and the blood supply of the tooth becomes impaired, and, thereafter, the blood supply is entirely destroyed, and death of the pulp results. The process is accompanied by a discharge of pus from the alveolus, and by a gradual disintegration of the alveolar process (caries). Finally, the tooth loses its alveolar and gingival connection and falls out.

On the loss of the tooth, the inflammatory symptoms immediately subside, a circumstance which, according to Marshall, lends great weight to the theory that the local manifestations of the disease are primarily associated with the dental tissues, and not with the gingivae or the alveolar processes. Witzel and others have maintained that the disease is primarily located in the alveolar border, and

that the gum and pericementum are involved as a secondary feature of the disease.

CASES IN ADULTS WITH AN UNERUPTED TOOTH

I have seen cases of osteomyelitis of the jaw, usually the lower jaw, with typical sequestration of portions of the alveolar processes or of the bodies of the bone in which the roentgen-ray studies demonstrated an unerupted tooth buried deeply in the bone tissue. The initial stages of the local condition are not accompanied by any apparent tooth infection, although, invariably, such an assumption is made as the patient interprets the pain as "toothache." Indeed, in one of my cases, the erupted teeth in the locality of the process were missing. No wound or other traumatic condition of the gum is present. The clinical course includes the usual manifestations up to and through sequestration, separation and exfoliation of the necrotic portions of the jaw. Subsequent closure of the wound or fistula does not occur until the unerupted tooth is operatively removed.

I interpret the sequence of events as follows: A hematogenous infection takes place in the bone tissue surrounding the cavity of the unerupted tooth, the latter possibly determining the point of fixation. Interference with the food supply of the unerupted tooth is a secondary phenomenon resulting from the involvement of the vascular channels of that portion of the jaw bone, itself affected by the inflammatory process. The unerupted tooth then acts as a foreign body, preventing healing of the wound until it is removed.

DENTO-ALVEOLAR ABSCESS

An important item in the development of osteomyelitic foci of either of the jaws is the suppuration which occurs. In this regard, all odontogenous considerations are of maximum importance. Suppuration about the teeth of purely dental origin is a very common occurrence. Following are the important facts to be remembered from the dental point of view in considering the subject of osteomyelitis of the jaws.

In dental practice, the term dento-alveolar abscess includes any accumulation of pus within a dental alveolus, or associated therewith. An acute and a chronic form of dento-alveolar abscess is described. In the acute form, the symptoms are often severe, with marked local and general constitutional manifestations. Extensive necrosis of the jaw bone proper and sloughing and gangrene of the soft tissues are frequent complications, and cases are on record wherein a general blood infection ensued, with an eventual fatality.

Bacillus pulpae-pyogenes of Miller is, according to Marshall, generally thought to be the organism responsible for the establishment of acute dento-alveolar abscess. It is found in decomposing and gangrenous pulps and in putrescent root canals, being exceedingly virulent. White mice inoculated with it by the peritoneal route die in from eighteen to twenty hours.

A chronic dento-alveolar abscess may be the sequel of an acute abscess which has established a fistula. A slow-developing chronic abscess also forms in the absence of any fistula formation. These are the so-called blind abscesses. The term is hardly a correct one, for the pus discharges through the pulp canal, which becomes its sinus, and it is therefore not a blind abscess. These abscesses are usually small, and, as a rule, are not very painful. In fact, all of the acute symptoms are greatly modified by reason of the ready exit of the pus from the beginning of the suppurative process. These conditions, if untreated, may persist for months or years

without causing any alarming objective symptoms. When a fistula exists, there is generally a constant discharge of pus into the mouth. The predominant micro-organism present is usually *Streptococcus viridans,* the organism which, according to Billings, Rosenow, Hartzell and others, seems to be the chief factor in the production of so many of the diseases which are now classified under the generic term "focal infection."

In the chronic form of abscess, the symptoms are usually much milder than in the acute variety, and there may even be a relative absence of symptoms as far as the local region is concerned. Rarely, exacerbation of infection occurs with secondary involvement and necrosis of bone and with the local and general manifestations of a local or general infection exactly as in the acute variety.

Most of the dento-alveolar abscesses either belong to the variety commonly called periapical or result as a further development of such an abscess. Periapical abscess is marked by an accumulation of pus about the apex of the root of a tooth and results in the following ways:

1. One type of periapical abscess results from the process by which pulp cavity and root canal involvement occurs following an initial superficial caries as described in the previous sections.

2. Periapical abscess forms after trauma, as after fracture passing through a tooth.

3. Periapical abscess is frequently found in the alveoli of vital teeth, as, for instance, in pyorrhea alveolaris and in inflammatory conditions arising from various forms of irritation and traumatism of the pericementum.

THE ASSOCIATION OF TRAUMA WITH DENTAL CONDITIONS

Trauma is associated with dental conditions in the following ways:

1. As a continual succession of individually inconsequential traumas necessarily taking place during eating. When combined with infection, caries of the teeth results.

2. As an isolated and extraordinary phenomenon, as, for instance, in the cracking of nuts or other hard objects with the teeth. It is undoubtedly true that, many times, this causes an injury to the apical artery or to its vascular connections and results in tearing of the vessel and in hemorrhage; such an injury is commonly asymptomatic; but the deprivation of blood supply causes death of the pulp, the tooth becoming insensitive. Infection from either an exogenous or a hematogenous source may be combined with this form of trauma, resulting in suppuration about the apex of the tooth, the characteristic of which is a foul odor.

3. In association with fracture of the jaw, the line of fracture may pass through the substance of one or more teeth so that a solution of continuity occurs between the crown and root of the given tooth. The further course of affairs is one of two: (*a*) If the fracture is of the simple variety, nothing may happen as far as the teeth are concerned and uneventful healing may result. (*b*) If, as is usually the case, the fracture is compound, and the line of fracture in the tooth is infected, as usually happens, the exposed pulp canal becomes infected and the infection spreads downward in the root canal toward the apex and results in suppuration. Commonly, the crown of the tooth is broken away and the root remains imbedded in the socket. Many dental surgeons have learned to extract root fragments of this kind in order to obviate any subsequent trouble.

In a comparison with infection in the bones (osteomyelitis), the following similarities are to be noted:

(*a*) In a general way, superficial caries of the teeth followed by extension of the process into the root canal corresponds in its mechanism to direct infection of bone tissue in general, following some form of trauma, as described in previous communications.[1] Naturally, in exposed structures such as the teeth, trauma need be of a relatively minimal kind, such as would ordinarily accompany the grinding of one tooth upon another in the mastication of food. A severer trauma accompanies the biting of hard food material (cracking of nuts), and frequently results in the breaking away of segments of the crown of the teeth. The further progression of the infection along paths produced by an eroding process parallels the further progression of an osteomyelitis of any bone under similar conditions. Naturally, too, the infection of the pulp following fracture of the tooth corresponds accurately to similar conditions in the bones of the body. This also is a form of primary infection.

(*b*) Suppurative conditions can establish themselves in the pulp cavity and root canal without direct external infection. The presence of pyogenic organisms in the blood current which may have gained an entrance through some abrasion of the skin or mucous membrane, or through a wound, or preexisting abscess, according to Marshall, has long been recognized. These are hematogenous forms of tooth infection. The guilty organisms are capable, when they become arrested in a dental blood vessel, of rapidly propagating and of establishing suppurative conditions. This explains the presence of abscesses of the pulp which are occasionally found in apparently perfectly sound teeth. Such dormant abscesses may not present any acute symptoms until they are opened, when, if the utmost aseptic precautions are not observed, they sometimes undergo most violent exacerbations of the inflammatory processes, which may terminate in the loss of the tooth and, in extreme cases, in establishing a fatal general blood infection. The clinical characteristics of teeth so infected include (1) an intact crown exterior and (2) an infected nonvital pulp.

In the absence of other demonstrable pathologic conditions in the given tooth, periapical abscess represents also, in my opinion, a metastatic infection following a pathogenesis similar to that in ordinary types of hematogenous osteomyelitis of other bones. The establishment of the given point of fixation depends on minor trauma incident to the grinding and mastication of food. The trauma serves to determine points where, because of tearing of the blood vessel and surrounding hemorrhage, resistance to infection is relatively less than elsewhere. Any bacteria circulating in the body circulation are likely to be blocked at these points where the circulation is interrupted. An inflammatory process develops and centers in and about the traumatized vascular channels present in the depths of the socket (the nutrient artery of the tooth). A relatively circumscribed lesion results, marked by erosion of the tip of the root and of the adjacent walls of the tooth socket, and by the formation of a minute abscess cavity lined by a granulation membrane.

Whether hematogenous periapical abscess of the teeth should not more correctly be classified as a chronic bone abscess of the jaw as has been described by Brodie in the long bones and in the jaws by Nussbaum, Frankel, Herzog and others is a nice point for discussion. As indicated previously, the blood supply of the individual teeth is furnished by termi-

1. Wilensky, A. O.: Ann. Surg., 82:781 (Nov.) 1925.

nal arteries, branches of the corresponding dental artery, which perforate the fangs of the teeth at their apices and penetrate the pulp cavity of the teeth. A thrombo-embolic process which becomes centered in these terminal branches would result in a small abscess immediately surrounding the vessel, which, because of the very nature of the anatomic arrangement, would be situated around the apex of the fang of the tooth; hence the name, periapical abscess. The association of this variety of dento-alveolar abscess with dental practice has apparently been established because of the proximity of the teeth; whereas, in truth, it might more correctly be classified with affections of the jaw.

PATHOGENIC RELATIONSHIP OF TOOTH INFECTION OR TRAUMA AND OSTEOMYELITIS OF THE JAW

Primary Infections.—Primary infections of the teeth necessarily follow injuries in which jaws and teeth are fractured simultaneously. Infection of the jaw bone proper is a simultaneous phenomenon in which the tooth infection is an accidental factor in the total lesion; and, vice versa, the jaw infection is an incident in the total lesion in which the tooth is implicated. A compound fracture is practically always present.

The line of communication with the exterior in the vast majority of the cases leads into the interior of the mouth through a rent in the mucous membrane. Contamination and infection occurs immediately; and the degree of the contamination and of resulting infection seems uncontrollable by any means at our command, inasmuch as it is impossible to (1) cleanse and debride the wound properly, (2) close the wound in the mucous membrane adequately and (3) prevent the continuous introduction of additional infection.

The essential pathologic process includes the introduction of infection into the opened vascular channels (thrombophlebitis) beginning in the plane and on either side of the line of fracture. The extent and intensity of the process depends on (1) the kind and virulence of the infecting organism and of the opposing antibody formation; (2) the extent of disturbance of the blood circulation, and (3) the character and degree of the consequent deprivation of food supply from the bone cells. The last item determines the amount of bone necrosis.

Hematogenous Infections.—In actual practice, hematogenous cases are seen by the dentists as the periapical variety of dento-alveolar abscess, easily distinguishable on the criteria presented by Marshall. To the surgeon, hematogenous cases of osteomyelitis of the jaws seldom present themselves with such a clear cut etiology. Usually by the time the patient comes in for treatment, the lesion is so extensive that it apparently implicates tooth and jaw and it becomes impossible to evaluate cause and effect except upon certain circumstantial evidence, such as (1) the presence of a suggestive or specific bacteriologic condition, (2) the association with an acute infectious disease or (3) certain clear cut pathologic conditions, such as the presence of an abscess deep in the bone tissue and isolated from the neighboring teeth. In most instances, the dental pathologic process seems to dominate the picture, and it is usually assumed that the latter is the cause of the entire lesion, whereas it is possible for a hematogenous infection to form points of fixation (1) either in the teeth or in the jaw, with subsequent extension of the infection from either one to the other; (2) in both the teeth and jaw simultaneously, or (3) in both the teeth and jaw at subsequent times, with coalescence of the

foci into a seemingly single lesion. An illustrative case follows:

An 8 year old boy was admitted to hospital with an alveolar abscess of the left mandible following the extraction of a tooth performed several days before admission. The temperature on admission was 104 F. and the patient appeared very toxic. There was marked swelling over the left side of the jaw, but no point of fluctuation. The temperature fluctuated between 103 and 105 F. In spite of apparent local improvement, the general condition became worse. Ten days later, the patient complained of pain in the right elbow, and a metastatic focus found in the lower end of the humerus was opened. Again there was improvement, but again the patient became toxic and went into stupor and coma. Blood cultivation revealed *Staphylococcus aureus*. The patient died two days later.

The clinical picture resembles closely that seen in cases of sepsis associated with the ordinary form of acute oestomyelitis.

In the usual clinical practice osteomyelitis of the jaw occurs because an inflammatory focus in intimate relation to the jaw bones spreads centrifugally along the connecting vascular paths by a process of thrombophlebitis until the osseous tissue itself is implicated and forms part of the total inflammatory lesion. This mechanism furnishes a large group of cases which have been termed extension cases of osteomyelitis.

Odontogenous Extension.—Cases of odontogenous origin are, of course, the commonest of the extension group. Nevertheless, spontaneous extension of the infection from a diseased tooth into the bone tissue of the jaw is comparatively uncommon, if one take cognizance of the total number of cases of dental infection. Anatomically, this would indicate that the pathologic process had extended in a retrograde fashion by a spread of the thrombosis into one or more of the larger branches, or into the main trunk of the dental artery (superior or inferior, depending on which jaw was involved) and had led to involvement of the blood supply and consequent necrosis and sequestration.

The cases which follow some operative (usually dental) trauma form the largest number of the cases in the extension group seen in ordinary practice.

The preexisting lesion in and from which the exacerbation and spreading of the infection originates is some variety of dental infection. Frequently, some form of dento-alveolar abscess is present, usually the simple periapical abscess; and the operative procedure varies from the cleaning out and repair by filling of cavities to tooth extraction and possibly other manipulations. Such cases are undoubtedly due to the mechanical spreading of infection along newly opened planes and their transmission into healthy vascular channels of the cortex and interior of the jaw bones. Secondary vascular thrombosis is always present and the extent of this determines the amount of devitalization and the extent of the consequent necrosis of bone.

Clinical History and Symptoms of Dental Postoperative Infection.—The usual clinical history includes the general and local symptoms associated with focal infection of one kind or another, or a frank toothache; a visit to the dentist during which usually extraction is done, and an almost immediate onset of pain in and swelling of the jaw, accompanied by chill, fever and other constitutional manifestations of a bacterial infection more or less virulent. The history frequently includes one or several additional operations in which incisions are made along the gums or externally, with little or, more usually, no relief. A characteristic manifestation is an exaggeration of the objective symptoms each time these additional incisions are made. Finally,

roentgen-ray studies reveal the presence of sequestrating bone and the diagnosis, perhaps suspected before that time, is established.

Extension cases of osteomyelitis of the jaws of any variety which occur after any type of dental or surgical operation or manipulation can only be considered such when the infection of the bone tissue is not a direct implantation during operative manipulation. True extension osteomyelitis would develop relatively slowly as a direct progressive growth of infecting bacteria along vascular or lymphatic channels into the bone tissue after the operation is completed, because bacteria are necessarily left in the field of operation when it is infected territory, and because new planes and areas of bone tissue have been exposed during the operation. Because the growth and progression of the bacteria are relatively slow, the clinical picture is proportionately insidious in its development and does not correspond to the more dramatic manifestations accompanying the direct primary infection of bone during any operative manipulation. Of course, this distinction is often most difficult to make clinically.

Another extension type of necrosis of the jaw of odontogenous origin which used to be seen many years ago is that which followed the injection of carbolic acid or the use of arsenic in certain lines of dental work. This practice has, very wisely, been given up by practically all dentists. I have the records of one case wherein the injection was made for the relief of pain. Extreme pain and swelling followed immediately and, after a very stormy time, in which necrosis of almost the entire alveolar process of the upper jaw resulted, a sequestrum was operatively extracted and healing ensued very slowly.

Blair and Brown report a case in a woman, aged 32 years in whose tooth some arsenic was left for two weeks. There was a local abscess, many teeth became loose and widespread jaw necrosis ensued. Several teeth extractions and sequestrotomies were necessary.

THE RELATION OF ODONTOGENOUS SUPPURATION TO SUPPURATION RESULTING FROM OSTEOMYELITIS OF THE JAWS

Knowledge of the forms and varieties of suppuration about an osteomyelitic focus in either of the jaws is important from the point of view of surgical treatment and, in this regard, a complete understanding of the odontogenous conditions is necessary. It appears that when a tooth is the seat of an acute dento-alveolar abscess, there is always considerable involvement of the bony structure surrounding the apex of the root. The pus is at first confined to the apical space, where it is surrounded by bony walls. As the pus accumulates, a rapid disintegration of the surrounding cancellated bone takes place, and a gradually increasing cavity is formed around the apex of the root. Such disintegration should not be classified as a form of osteomyelitis of the jaw unless secondary involvement with destruction of bone cells can be demonstrated in the jaw bone proper as a result of thrombophlebitic involvement of the vascular channels in the bone.

Classification.—The external or buccal wall of the alveolar process, being the thinnest, offers the least resistance to the progress of the constantly accumulating pus. For this reason, the abscess usually burrows through the external plate in its further development and points on the buccal aspect of the alveolar process opposite the apex of the root. Dento-alveolar abscesses can point in other ways after

penetrating the bony walls of the tooth socket:

1. Directly through the soft tissues. Dento-alveolar abscesses which point directly through the gum tissues are the most common. They rarely present any complications.

2. By separating the periosteum from the bone and forming a secondary pus pocket.

3. By following the pericementum along the side of the root and discharging at the margin of the gum. These are sometimes confounded with the so-called blind abscesses, but, in these cases, a careful examination will reveal the fact that there is no discharge through the pulp canal. Many times, this form of abscess is associated with pulpless teeth whose roots have been filled, but in which there is sufficient bacterial irritation to keep up a chronic discharge of pus through the sinus which has been formed by the side of the root.

4. Occasionally, toward the tongue. It is a curious fact that this direction of spread is not seen much oftener. It may happen with any of the previous groups. The amount of swelling in the floor of the mouth which follows this direction is extraordinarily large because of the looseness of texture of the tissues in this locality, and, unless one is on one's guard, the condition may be, as it has frequently been, mistaken for Ludwig's angina.

5. Through the floor of the nasal fossa. The relations of the roots of the incisor and cuspid teeth to the floor of the nose are such, in many instances, that appropriately placed dento-alveolar abscesses may point through the floor of the nasal fossa and produce a purulent discharge that might readily be mistaken for that of chronic nasal catarrh. The relation of the roots of the bicuspids and molar teeth to the floor of the antrum is such as to make it even less difficult for an abscess connected with one of these teeth to penetrate the floor of the antrum and give rise to an empyema.

Clinical and Objective Symptoms.—Clinically, as soon as the pus penetrates the bony walls of the abscess and escapes into the soft tissues, the severity of the pain is abated, but the tissues begin immediately to swell, and sometimes the swelling is very great. When associated with the upper jaw, it not infrequently closes the eye. When located in the lower jaw, in the region of the molars, it may be so extensive as to make deglutition impossible, and thus greatly obstruct breathing.

Clinical Course.—Acute dento-alveolar abscesses rarely heal spontaneously. There is a partial filling up of the pus cavity by the growth of granulation tissue but there is usually sufficient decomposition of tissue and growth of micro-organisms within the abscess cavity and the pulp canal to keep up a continuance of the suppurative process.

Occasionally, a dento-alveolar abscess may present chronic symptoms from the very beginning of the suppurative process, as, for instance, in blind abscesses. There is often a prolonged and obstinate irritation at the apical space and a persistent accumulation of pus, which finds an exit through the pulp canal, and the toxins of which are taken up and absorbed by the blood current or the lymphatic stream. These abscesses are among the most dangerous to the general health of the patient, as they are fruitful sources of general sepsis. Teeth so affected, and which do not respond to treatment after a fair trial, should be extracted.

Abscesses which have pointed through the external tissues of the face are nearly always of a chronic type, particularly

those which have had their origin in an early traumatism, perforation of the cementum, breaking of an instrument within the root canal or impaction of a tooth.

Association of Odontogenous Conditions and Jaw Involvement.—It should be assumed in cases of osteomyelitis of the jaws associated with odontogenous factors that any suppuration beyond the simplest forms is to be ascribed to the associated lesion in the jaw bone. In going through the various dental contributions, one notes how frequently the suppuration described is said to be complicated by necrosis of portions of the jaw bone. Any suggestion of necrosis of the jaw should, of course, remove such suppuration from the category of dental to that of the osteomyelitis of jaw bone proper, even though the exfoliation of a sequestrum is not always necessary to prove bone involvement, because these are essentially subperiosteal abscesses, which are not always associated with necrotic bone.

The actual mechanism by which a dental infection is transmitted to the jaw bone proper is thrombophlebitis. The vascular lesion is usually also the major component of the dental infection itself and any spread of this in a retrograde fashion into any of the radicles of the dental vessels becomes, in due course, either rapidly or more slowly metamorphosed into an acute inflammation of bone with all of its consequences.

(Any further consideration of osteomyelitis of the jaws proper is beyond the scope of this paper. A complete exposition of the subject, including suppuration and necrosis and sequestration accompanying osteomyelitis of the jaws, is published elsewhere.[2])

2. Wilensky, A. O.: Arch. Surg., 25:183 (July) 1932.

COMPLICATIONS

General Infection.—In dental practice, a general infection is almost invariably associated with some form of dentoalveolar abscess usually of the periapical variety. Acute and chronic forms of "septicemia" are described (Marshall, Thibault and Raison, etc). The chronic form corresponds to one of the clinical entities, referred to usually under the general term of "focal infection" in this country, and is of no interest to us in relation to the subject under discussion. The acute form of general infection is the one which interests us. According to Marshall, this has been seen in dental practice in association with periapical abscess.

In surgical practice, the clinical course is different. Almost invariably, the substance of the jaw bone proper becomes infected and a severe form of osteomyelitis results, developing coincidently with the general infection. Forms are described in which a general infection occurs as a sudden acute overwhelming exacerbation of a previously existing focal infection, usually a dormant periapical abscess, and without intervention of operative or other form of trauma. These forms are unusual. Much more commonly, the general infection is associated with some operative manipulation indicated by some dental condition. The following two groups are met in clinical practice:

1. Following some operative manipulation—extraction of a tooth, treatment of periapical abscess, etc.—it is uncommon to have a high postoperative fever with corresponding subjective and objective symptoms. Undoubtedly, excessive temperature of this kind must be referred to a transient bacteriemia with corresponding states of general infection of similar evanescence. Commonly, the

cases in this group go on to complete retrogression and recovery as regards the general and local condition.

2. There are a number of cases of general infection which, originating as do the cases in the previous group, go on and show a steady progression even to fatality. The frequency of these latter cases is not correctly appreciated by the profession. I usually see one or two of such cases in hospital and private practice each year. General infections of this type, of dental origin and of operative mechanism, are apt to be of intensity and marked malignancy, to be complicated by intracranial complications and to end fatally within a few days.

General infection occurring as a late phenomenon in the course of an osteomyelitis of the jaw follows some form of trauma, either that occurring during a dressing of the wound or following some operative intervention. Clinically, general infection at any late stage of the disease, under the conditions specified, follows the form of development indicated in the immediately preceding two paragraphs and with similiar consequences.

Under any of these conditions, complicating nearby or distant lesions occur as a result of the transmission of the infecting bacteria through the blood stream. Some of these possible secondary conditions are also capable of being associated with a general infection. Commonly one is faced with a combination of conditions in which the general infection can be referable to either the original jaw infection or to complications, as, for instance, a cavernous sinus thrombosis or a meningitis. Positive and negative blood cultures may be obtained in otherwise equally severe and fatal cases. The significance of positive and negative blood cultures in clinical practice in cases of acute infection has been discussed elsewhere.[2]

Associated Conditions of Hematogenous Origin.—In hematogenous cases and in those in which secondary general infections take place, metastatic foci can and do develop as a result of bacteriemia. Indeed, in some of the hematogenous cases, the jaw lesion itself may be only one of many manifestations. These include (1) other bone lesions; (2) suppurative and nonsuppurative forms of arthritis; (3) infections of the various body cavities; (4) forms of endocarditis and pericarditis; (5) lung abscesses; (6) renal infarcts, and (7) minor abscesses in the various fascial planes. These are common to all forms of hematogenous infections and further discussion of them is beyond the scope of this paper.

Local Complications.—A group of complications occur as a result of local extension of the disease. These include:

1. Various abscess formations, including orbital abscess and empyema of the antrum. Rather uncommon complications which result from the pointing and rupture of the cortical forms of abscess include those described in the literature in which rupture took place into the larynx or trachea, or those which extended into the thorax.

2. Ankylosis of the temporomaxillary articulation occurs as a result of involvement of the glenoid process in the osteomyelitic lesion, or as a result of calcification of the muscular and tendinous tissues about the joint and upper part of the ascending ramus. This includes various forms of arthritis of the temporomaxillary articulation.

3. Lesions about the angle and body of the lower jaw, which can rarely cause a phlebitis of the facial vein radicles, but which, when once established, have a predeliction for spreading upward into the cavernous sinus. I have the records

of one private case and have seen one case in hospital practice and one report in the literature, by Coppens:

The patient was 21 years old and a woodworker. Except for a fracture of the arm and an attack of typhoid fever many years before, he had never been ill. Fifteen days before admission, he became suddenly ill with excruciating pain in the left lower jaw, with high fever, delirium and other signs of a general infection. Incisions were made in the gingivolabial fold, which gave little relief and revealed a process in the lower jaw. The illness was progressive thereafter until the patient died.

The postmortem examination revealed an entire denudation of the entire left lower jaw. The bone was necrotic and gangrenous. The process seemed to stop anteriorly at the median line, and to extend up to the temporomaxillary articulation. All the veins which have their point of origin in the inferior maxilla were full of pus; the external jugular vein was full of pus, and the pus was traced up into the left cavernous sinus. The right side was healthy. A meningitis was also present.

In another case of which I have knowledge, the same sequence of events followed an infection in one of the molar teeth of the upper jaw.

4. Meningitis, a very rare complication. Coppens' case presented this condition.

5. Various forms of encephalitis. This condition not uncommonly complicates accessory sinus disease. When sinus disease (empyema of the antrum of Highmore, usually with and sometimes without involvement of the major portion of the frontal, ethymoidal and sphenoidal sinuses) complicates osteomyelitis of the upper jaw, encephalitis appears as a complication. Halloran's case was an example of this:

The patient had osteomyelitis of the upper jaw with empyema of the maxillary sinus and roentgen-ray evidence of pansinusitis. After drainage was carried out, the patient left the hospital in a much improved condition, only to return five weeks later with a progressive development of bilateral labyrinthine symptoms. At the peak of development, there were bilateral paralysis of the eighth nerves, and paralysis of the left seventh and of the left twelfth nerves. The laboratory tests were essentially negative. Improvement occurred in the manifestations referable to the seventh nerve. Paralysis of the eighth nerves was still persistent at the time the case was reported.

6. Cerebral abscess. Marchand mentions cases of cerebral abscess having been reported as complicating osteomyelitis of the jaw. Cerebral abscess under these conditions represents a further development of an encephalitis. In common with other wounds, wounds resulting from osteomyelitis of the jaws may be complicated by erysipelas and by tetanus.

TREATMENT

For purposes of treatment, cases of combined dental infection and osteomyelitis of the jaws can be divided into the following groups:

1. Cases with clinical signs of a general infection.

2. Cases with little or no signs of a general infection, but with various grades of the local lesion.

3. Cases with complications. (The discussion of the treatment of any of the complications, except that of local abscess formations is not included.)

Treatment of Cases with Clinical Signs of a General Infection.—Fulminant forms of general infection are usually of such a high order of severity as to render useless any form of local or general treatment. The group includes those cases of very sick patients with high fever and other clinical signs of general infection, but in whom culture of the peripheral blood is usually negative. Spontaneous retrogression of the infection is common in this group and occasionally as a recognizable partial or complete result of the form of treatment employed.

A fatal outcome may occur subsequently either because of an intensification of the infection and a change from a sterile condition of the blood to a bacteriemia, or because of the occurrence of local or distant complications. The cases in this group include some of the severer cases of osteomyelitis following trauma and fracture of the jaw and teeth, and cases of acute infection of the jaw following operative manipulation. In these cases, treatment should be directed persistently to the local condition, as detailed subsequently.

Treatment of Cases with Little or No Sign of General Infection.—Cases in this group consist of the majority of those seen in practice in which the problem is that of the local lesion and its local complications, principally abscess formation, and into which the question of general infection does not enter. Treatment resolves itself to the best ways and means which will lead to the minimum loss of tissue, the most rapid exfoliation of the sequestra, the most rapid healing of the wounds, the greatest conservation of form and function and the best general advantage to the patient.

Common accumulated experience and the literature make it evident that, for the local lesion, the general practice of conservation is the accepted principle of treatment at the present time. This is in accord with the theoretical considerations of the subject which I have given on several different occasions. In upper jaw lesions, conservatism has been forced on the surgeon because of local anatomic and technical considerations. In the lower jaw lesions, conservatism is based on experience.

Ordinarily, the treatment of the local lesion itself should be considered under the following headings: (1) prophylaxis, including the treatment of any antecedent condition; (2) treatment of the local condition in the early stages, and (3) treatment in the later stages.

Effective treatment of the antecedent condition is necessary when the osteomyelitis follows some form of trauma and when it is associated with an odontogenous condition. It is beyond the scope of this communication to consider the traumatic conditions of the jaw (fractures, gunshot wounds, operative procedures, etc.,) which are complicated by osteomyelitis.

The odontogenous conditions are important. Adequate attention to them includes certain prophylactic measures.

Prophylaxis is the most important single item in the prevention of any local infection of the jaw bones from odontogenous sources. Generally speaking, the best that one can do as regards any form of general infection is also of a preventive nature purely. Most of the cases in practice are in association with odontogenous conditions, occurring as a result of unwise dental procedures or as unexpected consequences of apparently necessary manipulation, even in very capable hands. The most important means of prevention lie in the field of dental prophylaxis, which would tend to avoid, to decrease in numbers or to wipe out completely any form of dental infection. The importance of this simple preventive means cannot be overemphasized, and fortunately it is being recognized more and more throughout the country. The next most important means of prevention of local or general infection lies in closer attention to the rules of asepsis in dental practice among the rank and file of the dental profession and keener appreciation of the great importance of gentleness in all dental procedures. For practical purposes, this is all that one can do, especially in cases of general infection because, when infection has occurred, care and healing depend on the relationship of individual

resistance and the virulence and intensity of infecting organism; and over this interaction of natural forces, medical knowledge has little control at present.

Cases are well known, especially to the dental profession, in which apparently spontaneous exacerbation of infection extending into the tissues of the jaw bones occurs in previously existing periapical forms of dento-alveolar abscess. Efficient prophylaxis would prevent these accidents also except in those cases in which the periapical abscess is itself evidently a manifestation of a hematogenous infection.

In actual practice, infection of the jaws occurs as a complication of the following dental procedures:

1. Extraction of teeth. It is common practice among the dental profession to pack the tooth sockets after an extraction. This one procedure accounts for a large proportion, if not the vast majority, of infections of the jaw, inasmuch as the corking up of the secretions in the cavity of the teeth socket practically forces the infection into the bone tissue of the jaw. The dental profession should avoid this practice and should accept the policy of nonpacking after extraction as an important prophylactic measure.

2. Treatment of dento-alveolar abscess of purely odontogenous extent and limitations. This, also, is a prolific source of infection of the jaw bone. Many times, infection of the jaw occurs because of the mere presence and the nature of the infecting organism and the lack of general, and especially local, resistance of the patient and the tissues. But this is equally often aided materially by the lack of aseptic technic and by the lack of gentleness in manipulation. For periapical abscess, extraction of the teeth is the simplest and most common treatment. Naturally, the prophylactic considerations detailed in the previous paragraph are applicable with increased force when infection is present.

The majority of periapically infected teeth do not require anything further than a clean extraction. Curettement through the socket of the extracted tooth appears to me not only questionable but also totally inadequate. The apical contents, by such a procedure, are usually thoroughly macerated with the normal blood clot and remain in the socket, thereby creating an excellent culture medium. The bone curet in the hands of an eager, unskilled operator is a dangerous instrument, especially when that operator feels called on to curet all sockets after extracting whether the case is an acute alveolar abscess or not. Many cases of osteomyelitis of the underlying jaw are the result of this form of treatment.

When once an acute osteomyelitis has established itself in the bony tissues, it is important to recognize that, in the early stages, a period of extraordinary virulence of infection is present, usually lasting a very few days and in which any active or radical measures are likely to be productive of much harm, especially in the spread of the local infection, and possibly even, as pointed out previously, in the production of a general infection. The importance of absolute conservatism throughout this stage is paramount.

In the presence of odontogenous conditions, no extraction should be made at this stage, nor should any other dental procedure be carried out. In the presence of any preceding trauma or operation, due regard should be had of the danger of causing an increased exacerbation of the infection. Certainly, under such conditions, no further operative procedure should be undertaken; and any absolutely necessary dressings of the wound should be carried out most gently and with knowledge of the danger well in mind. During this stage of increased virulence,

only the simplest measures necessary to proper cleanliness of the mouth should be practiced; and the contraindication to extraction or further manipulation should be absolute.

Benevolent change from the period of increased virulence is manifested by a lessening of the swelling of the tissues of and about the jaws, and by a perceptible decrease in the general and other local manifestation of bacterial infection. Any dental condition which needs correction should be given attention at this time and, in actual practice, this correction should be strictly limited to the extraction of definitely diseased teeth from which the local lesion apparently originates. The value and importance of the extraction lies in the fact that removal of the diseased tooth allows free drainage from any associated form of dento-alveolar abscess; and, by not doing anything else, and especially by leaving the socket free from packing, it can be confidently expected that the odontogenous lesion will quickly and progressively retrogress. In cases of other etiology, especially operative cases, the policy of conservative passivity should be persevered in during this stage also.

In hematogenous infection of the jaws, the policy of conservatism during the early stages of development of the jaw lesion seems to me to be also of extreme importance. In these cases, the general principles underlying the treatment of acute osteomyelitis in general hold good, and, as so often happens in osteomyelitis of the irregular bones, this attitude is fortified and made imperative by anatomic and local technical considerations.

Sooner or later in any of the varieties of acute osteomyelitis of the jaws, suppuration occurs. The abscesses[2] are situated in relation to the upper and lower jaws and to the teeth from which any osteomyelitis of the jaws had originated. They vary in size from small insignificant lesions to those of great size and clinical importance.

Commonly, any of these abscesses is associated with much induration in the tissues in and about the jaw. The emptying of the abscess leaves an extensive swelling which slowly and spontaneously disappears, the more quickly when poulticed. It is important to remember that no attempt should be made at the time the abscess is opened to execute any procedure on the bone itself. Usually, any such attempt is followed by increased swelling and induration, if by nothing more dangerous.

The late stages of acute osteomyelitis of the jaws is characterized as the period in which sequestration occurs. (The causes for this and the physical and anatomic characteristics of the various forms of necrosis have been detailed in another communication.[2]) Efficient separation of the necrotic portions of the bone usually occupies from thirty to ninety days, depending on the area of necrosis and other local conditions. During this period, suppuration is apt to be profuse both through the external sinuses and through the sinuses in the oral cavity about the teeth. Retention of pus is common and new closed off pockets may and do commonly form, requiring additional incisions. It is important to remember that, during these procedures, the aim should be to restore or create drainage for the accumulated pus, and on no account should there be any interference with the bony structure. The reasons for these considerations include: 1. An inability to recognize and distinguish between necrotic and healthy viable bone except through the plane of separation between healthy and dead bone. 2. The fact that sequestration, especially in the cases of more moderate extent, either does not occur, or occurs in such small or limited extent as to be negligible; and when it is

of such small or limited, extent, nature will take care of and properly exfoliate such sequestra very much better and with less damage than can the surgeon. In many of these cases, the pathologic process is that of a subperiosteal abscess pure and simple. 3. The fact that when appreciable areas of bone necrosis do occur, these will be of minimal size, commensurate with the position of the thrombophlebitic lesion and of its consequent disturbance of blood supply, when the natural process of sequestration is allowed to go on undisturbed. 4. The fact that if interference with the bone structure is practiced at too early a stage, it most commonly results in an increase in size of the lesion and of the area of necrosis. 5. The fact that, in the lower jaw, the importance of waiting is paramount for the purpose of allowing sufficient involucrum to form in order that the form and configuration of the lower jaw and the chin may be retained. In the upper jaw, this consideration does not exist, inasmuch as involucrum does not form.

These are the considerations which decide the advisability of delaying sequestrotomy until conditions are most favorable, that no untoward consequences may follow and that conservation of form and function may be possible.

Under such restrictions and conditions, the process of sequestration and unaided or operatively aided exfoliation are reduced to the simplest form. Many times, the sequestrum will be spontaneously exfoliated; or, during one of the dressings, a sequestrum presenting in the mouth of one of the sinuses can be easily withdrawn, almost without the patient's knowledge. Healing then proceeds quickly. Both of these methods of termination are common in an acute osteomyelitis of the jaw.

When an operative procedure is necessary for the removal of the sequestrum, the following simple rules should be remembered: 1. Whenever possible, and to as large an extent as possible, the operation should be performed through the oral cavity. 2. Enlargement of any preexisting wound should be commensurate with the size of the sequestrum. 3. Chiseling away of any involucrum for this purpose should be minimal and should be so planned that the least possible deformity results. As a rule, very little chiseling is necessary. As was indicated previously, this applies practically exclusively to the lower jaw. 4. Any manipulation through the oral cavity or from the outside should be done in such fashion that a minimum amount of trauma results to the muscular and tendinous tissues about the condylar and glenoid processes of the mandible, in order to prevent any calcification of these tissues and a consequent ankylosis of the temporomaxillary articulation. 5. Any external incision should be planned with due regard to a minimal resulting visible scar. 6. Any necessary drainage should be instituted as far as possible from the inside of the mouth and, in the largest number of cases, this can be done exclusively. Then one can neglect any open sinuses which develop externally, and it usually follows that the external openings promptly close.

Healing after secondary operations is usually a protracted affair and the average case extends over a period of three or four months before complete closure is secured. Occasionally, further measures are necessary and, during these procedures, much the same rules should be followed. The important rule to remember is that the less one is forced to do, the less damage one is apt to do, the better for the local condition and the quicker the subsequent healing.

Fry calls attention to the frequency with which pathologic fracture occurs in

osteomyelitis of the mandible. This has not been my experience. The diagnosis is made on the basis of deviation of the mandible from the midline to the affected side, crepitus and the roentgen-ray findings. The amount of displacement depends on the line of fracture, the presence of teeth on the smaller fragment, with occluding teeth on the maxilla, and the amount of loss of bone. The general principles underlying the treatment are correct alinement, reduction of sepsis and immobilization of the fragments.

Dental splints should be fitted as early as possible to immobilize the fragments and thereby relieve the pain, reduce the danger of sepsis and allow the patient to take food more easily. Early surgical intervention by wiring or plating is contraindicated. Reduction of sepsis may be accomplished by constant irrigation, efficient drainage and the removal of septic teeth and teeth in the line of fracture. Splinting is used to aid in reduction of the displacement and to immoblize the parts. Immobilization is obtained by means of bandages and external supports, interdental wiring or the use of dental splints.

In upper jaw cases, even extensive necrosis does not necessarily mean extensive deformity, and one is constantly surprised at the little change that is perceptible in the contour of the cheek and face. When the antrum has been exposed, healing is accompanied by epithelization of the cavity, and the opening contracts but little and is permanent. It is necessary to have an obturating appliance made to close the opening and the obturator can be made to carry artificial teeth to replace those missing. Symptoms referable to the orbit disappear with the subsidence of the process and the healing of the lesion. Nasal symptoms usually need local attention for a little time, especially when, as so often happens, they are associated with a disturbance in the antrum.

In lower jaw cases the presence of the involucrum results in an appreciable thickening of the jaw. The amount of deformity is little. Dental appliances are necessary to make good the loss of teeth which have been destroyed by the disease or as a consequence of operative manipulation.

When large portions of the jaw are lost, the remaining fragments are drawn toward each other by the contraction of the muscles and the teeth are thrown out of occlusion, in many cases great deformity resulting, especially when the chin is swung to one side. To prevent this, it is necessary to keep the remaining portions of the jaw immobile and in proper alinement by fixation of the teeth in occlusion. The simplest method is interdental ligation. Sometimes, because teeth are missing, a splint may be necessary, or even a vulcanite bar may be placed in the wound to maintain contour until new bone has formed. This bar can be removed and cleaned, being also gradually reduced in size as new bone is formed.

MINERAL METABOLISM IN RELATION TO DENTITION. II. BASE-FORMING AND SALT-POOR DIETS*

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IN the first paper of this series,[1] the relationship of heredity, prenatal conditions and infant nutrition to mineral metabolism was given consideration. In this article, the inorganic mineral requirements of the human body and the effect on mineral metabolism of certain types of diets will be considered.

TABLE 1.—MINERAL ELEMENTS IN THE HUMAN BODY

Element	Amount in Man Weighing 70 Kg. Gm.
Calcium	1,050.000
Phosphorus	700.000
Potassium	245.000
Sulphur	175.000
Sodium	105.000
Chlorine	105.000
Magnesium	35.000
Iron	2.800
Iodine	0.028
Fluorine	Minute quantities
Silicon	Minute quantities
Manganese	Minute quantities

The ash of the human body furnishes a natural basis for a study of the residual inorganic requirements of the organism. The amounts of inorganic mineral elements composing this ash as given by Rose[2] are listed in Table 1.

As will be seen, a man weighing 70 kg. or 150 pounds contains about 1 kg., or one-seventieth of his weight, in calcium, 0.7 kg. of phosphorus and smaller amounts of the other elements. The amount of sodium, 105 gm., exactly balances the chlorine present; a point to which I shall refer later.

The need of the minerals found in major quantities, such as calcium and phosphorus, has long been recognized. The ones required in minor amounts, including potassium, sulphur, sodium, chlorine, magnesium, iron and iodine, and those needed in minute amounts, "trace" elements,[3] are receiving more serious consideration than formerly. Until recently, when mere traces of minerals were found in the ash of the human body, or in one of its organs, they were considered accidental or inconsequential. Mendel's so-called "little things"[4] in nutrition are assuming new importance. One of these, copper, is now recognized as absolutely

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1. Moore, C. U.: Mineral Metabolism in Relation to Nutrition, 1. Some Prenatal and Postnatal Factors, J. Nebraska D. Soc., 9:6 (Sept.) 1932.

2. Rose, Mary S.: Fundamentals of Nutrition, New York: Macmillan Co., 1927, p. 147.

3. Rose, Mary S.: Nutritional Significance of Some Mineral Elements Occurring in Traces in Animal Body, Yale J. Biol. & Med., 4:499 (March) 1932.

4. Mendel, L. B.: Nutrition; Chemistry of Life, New Haven: Yale University Press, 1923, Chap. 11.

essential in the prevention and cure of anemia. The need of iodine for the prevention of goiter is well recognized. Even this condition may revert to a calcium deficiency, for, as Scott[5] points out, the endocrine glands do not function properly if the calcium metabolism is out of balance.

Since the body contains nearly as much of calcium as of all the other minerals combined, it is evident that a large daily intake of this mineral is necessary. From the volumes written on calcium metabolism and its relation to rickets, osteomalacia, tetany, dental caries, etc., one would be led to suppose that the daily needs of a normal individual are definitely known; but the literature contains such varied statements that no doubt McCollum[6] is correct when he says that "the optimum amount of calcium in the diet is still unknown." A quantitative study of the minerals ingested compared with those excreted establishes to a certain extent the amounts required by the organism. The work of Sherman,[7] Lusk,[8] Rose[2] and others has shown that the daily diet should contain certain important minerals in approximately the amounts given in Table 2.

TABLE 2.—MINERAL ELEMENTS NEEDED DAILY BY ADULTS*

Elements	Amount
	Gm.
Calcium	1.000
Phosphorus	0.88
Iron	0.015
Iodine	0.005 (per year)

*Children need 50 per cent more.

Time forbids an intensive consideration of all the minerals needed in the human diet. The versatility of the body in using what our fads or fancies choose to give for its sustenance is partly due to its storage supply of so-called buffer substances. For example, the extra oxygen needed by the muscles when a person runs to catch a street car could not be supplied by the lungs fast enough to prevent one's falling in a faint. The red blood cells are the buffers or the storage batteries which supply the extra oxygen required during such an emergency.

The maintenance of a proper mineral balance in the body requires mineral buffers, such as the phosphates, to prevent an excessive accumulation of unneutralized acids. Shohl[9] states that a normal man's daily requirements of alkaline elements approximates 150 c.c. of tenth-normal solution.

For some years, dental caries has been attributed to oral acidity, and in an effort to overcome this condition, alkaline dentifrices have been created. These may temporarily counteract the effect of acid mouth, but they do not correct the constitutional cause of the trouble. Bödecker[10] asserts that tooth resistance to decay is dependent on the dental lymph, and this will, if adequately alkaline, neutralize the lactic acid formed by bacteria in the mouth. On the other hand, Hoppert, Webber and Canniff,[11] of the

5. Scott, G. D.: Tooth Decay and Balanced Metabolism in Infancy and Childhood, M. J. & Rec., 125:242 (Feb. 16), 387 (March 2) 1927.

6. McCollum, E. V.: Newer Knowledge of Nutrition, New York: Macmillan Co., 1925, pp. 360, 477.

7. Sherman, H. C.: Chemistry of Food and Nutrition, Ed. 3, New York: Macmillan Co., 1927, p. 310.

8. Lusk, Graham: Science of Nutrition, Philadelphia: W. B. Saunders, 1928, p. 482.

9. Shohl, A. T.: Mineral Metabolism in Relation to Acid-Base Equilibrium, Physiol. Rev., 3:509 (Oct.) 1923.

10. Bödecker, C. F.: New Theory as to Cause of Dental Caries, D. Cosmos, 71:586 (June) 1929.

11. Hoppert, C. A.; Webber, P. A., and Canniff, T. L.: Production of Dental Caries in Rats Fed Adequate Diet, Science, 74:77 (July) 1931; J. D. Res., 12:161 (Feb.) 1932.

University of Michigan, have recently produced caries in rats by feeding cornmeal, which easily becomes impacted in the teeth, but were able to prevent caries by the substitution of oatmeal, which does not become impacted.

As their diets contained 60 per cent or more of cereal, the metabolic effect within the organism would probably be acid. It is generally considered that a properly balanced diet should be neutral or alkaline in its metabolic effect.

ACID-BASE BALANCE

The chemical reaction of a food may be determined by the ash remaining when the food is burned. This occurs whether the burning is done outside the body or in the muscles. The ash left may be acid, neutral or alkaline, according to the kind of food consumed. A concise classification of ordinary foods divided according to this standard is given in Table 3.

TABLE 3.—FOODS GROUPED ACCORDING TO ASH REACTION

Acid	Neutral	Alkaline
Cereals	Vegetable Oils	Vegetables
Meats	Butter	Fruits
	Lard	Milk
Eggs		Most nuts
Coffee	Sugar	Potatoes
Tea		

A diet unbalanced on the side of either alkaline-ash or acid-ash foods is undesirable. As Henderson[12] succinctly states, neutrality is a definite, fundamental and important characteristic of the organism. If one analyzes his own daily diet as Manville[13] suggests, does he not find that he consumes more calories of the acid-ash foods, such as meat, eggs, fish, chicken, bread and cereals, than of the alkaline-ash ones? Hence, we see how little danger there is of erring in the direction of too alkaline a diet. The overconsumption of acid-ash foods is very common, and the danger therefrom very grave. In addition to the acids accumulated in the body from this type of food, the products of catabolism of body tissues are acid in reaction. An excess of base-forming elements in the diet is therefore needed to produce neutrality and to prevent excessive formation of acid ions. Otherwise, an acid-ash type of acidosis develops, with headache, nausea and vomiting as acute symptoms, or mouth acidity, stomach acidity, acid perspiration, etc., as chronic results. Manufacturers say that acid perspiration often causes rapid deterioration of good silk hose.

It is a commonly accepted fact that the taste of a food is no indication of its acid or alkaline activity in the system. Citrus fruits have a highly acid taste, but a marked alkaline effect on the body; while bread and meat, neutral in taste, have an acid effect. This alkaline effect of fruits results when their organic acids are burned to carbon dioxide, which is eliminated by the lungs, the nonrespirable organic bases remaining producing an alkaline metabolic effect.

The correction of hyperacidity in the system can be accomplished by an adequate consumption of milk, fruits, all vegetables, except lentils and sorrel, and most nuts, except peanuts and walnuts. Irish potatoes vindicate their right to a daily appearance on our tables because of their alkaline effect in the system. Most cooks consider rice, hominy and macaroni as proper and adequate substitutes for potatoes because of their common element, starch. In point of fact, they are the op-

12. Henderson, L. J.: Regulation of Neutrality in Animal Body, Science, 37:389, 1913 (also quoted by Sherman, H. C.: Chemistry of Food and Nutrition, Ed. 3, New York: Macmillan Co., 1927, p. 289.)

13. Manville, I. A.: Personal communication to author. Manville, I. A., and Winchell, R.: Food Tables for Use in Acid-Base Diets, Northwest Med., 31:464 (Oct.) 1932.

posite to potatoes in their effect in the system, as they are all three acid formers,[14] while potato is a base-forming food.

Sometimes, an alkaline food may be changed into an acid one by prolonged cooking, which dissolves out the alkalis anl leaves an acid-producing residue, as shown by Berg.[15] Foods are also affected by the kind of soil in which they are grown, and the fertilizers used. Elvehjem and Hart[16] found that the copper content of lettuce, for instance, can be increased 148 per cent by adding 500 pounds per acre of crystalline copper sulphate. Canned foods are, of course, affected by the preservatives used, such as boric, benzoic and salicylic acids. These, even in very small amounts, cause foods to have a surplus of acid ions.

DETERMINATION OF THE BIOLOGIC EFFECT OF A DIET

There are three ways of evaluating the chemical reaction of foods: (1) by taste; (2) by ash-value, and (3) by biologic effect. The first of these we have shown to be unreliable. Even the second, the determining of the ash-value of a food by burning outside the body, is not wholly reliable. Ullman[17] credits Kroetz with introducing the conception that the ash-value of a food cannot be relied on entirely to give a true picture of its metabolic effects. The metabolic value of a food can be determined only by what Kroetz[18] calls its biologic effect on the human body. Fortunately, the nonrespirable ash of our foods is eliminated from the body chiefly by the kidneys. Blatherwick,[14] in 1914, demonstrated the specific influence of the ash of foods on the composition of the urine, showing that the consumption of sufficient base-forming foods produces alkalinity of the urine; which becomes, therefore, an excellent means of determining the true biologic effect of the food consumed, and hence of studying the mineral balance of the human body. The acid or alkaline reaction of the urine can be determined roughly by the use of litmus. This test should give a neutral reaction except after violent exercise, when large amounts of acid ions are thrown out. If the litmus test shows acidity, a more careful study should be made and the diet corrected by reducing the consumption of acid-ash foods.

The determination of the degree of acidity or alkalinity of the body fluids, such as blood and urine, is measured most accurately by their hydrogen ion concentration, or p_H. The neutral point, p_H 7, is found, as you will recall, in distilled water. A decinormal solution of hydrochloric acid has a p_H of 1; a hundredth normal, of 2; a millionth normal, of 6. The living body is always slightly alkaline, that is, it keeps itself a little above a p_H of 7. The blood has a hydrogen ion concentration normally maintained at p_H 7.4. If the p_H of the blood is altered 0.4 of a point in either direction, death occurs. If it rises 0.4 of a point, convulsions precede death; if it falls an equal amount, coma and death occur. Lesser changes produce disease symptoms that vary with the amount and direction of

14. Blatherwick, N. R.: Specific Rôle of Foods in Relation to Composition of the Urine, Arch. Int. Med., 14:409, 1914.

15. Berg, R.: Grundlagen einer richtigen Ernahrung, Ed. 7, Dresden, 1930.

16. Elvehjem, C. A., and Hart, E. B.: Proper Content of Feedingstuffs, J. Biol. Chem., 82:473 (May) 1929.

17. Ullman, E. V.: Chronic Sinus Diseases; Their Dietetic Treatment, Northwest Med., 31:240 (May) 1932.

18. Kroetz, C.: Theoretishche und Praktische Grundlagen der Diätbehandlung mit Sauren und alkalishen Kostformen., München. Med. Wchnschr., 76:1788 (Oct. 25), 1842 (Nov. 1) 1929.

the change. Estimation of the hydrogen ion concentration of the urine can be made quickly by colorimetric methods using the Heilege-Klett or La Motte apparatus.[19] Any laboratory technician or nurse can learn to perform these tests easily and accurately. If the urine is kept close to the neutral point, p_H 7, the metabolism of the body is operating with the least amount of friction, i. e., the most economically. Even the neutral point (p_H 7) indicates, according to Sansum[20] that the urine is twice as acid as the blood. In some persons, the urine burns the mucus membranes and skin when it becomes 100 times as acid as the blood, p_H 5.4, while others have no such indicative sensation with urine 1,000 times as acid as the blood, p_H 4.4.

Hess[21] has shown that a diet to which hydrochloric acid is added produces rickets. Conversely, in 1924, Martha Jones[22] and her co-workers at the University of California found that a highly alkaline diet produced rickets. Apparently, neither of these workers tested the effect of the diet used on the p_H of the urine. In 1921, Hodgson[23] pointed out that there is an increased acidity of the urine in rickets. This important observation was confirmed in 1927 by György of Heidelberg,[24] who found also that treatment with cod liver oil or with ultraviolet light usually brought about a decrease in the amount of acid excreted in the urine.

Blatherwick and Long[25] demonstrated that the consumption of a considerable quantity of orange juice produces an alkaline urine, and that similarly large amounts of lactic acid milk caused marked acidity of the urine. This may well be remembered when we add lactic acid to infant feeding formulas.

Bunting[26] and his co-workers at the University of Michigan produced dental caries in rats by feeding "a considerable amount of sugar." In their survey of a group of children fed over a period of two years on diets containing a minimum of sugar and of white flour, a quart of milk, some green vegetables and fruits, the development of caries was greatly reduced. Their menus indicate that the food ash would be alkaline. When Hanke[27] recommends a pint of orange juice a day for its vitamin C content, he is undoubtedly getting part of his good results from the base-forming value of

19. Osgood, E. E., and Haskins, H. D.: Laboratory Diagnosis, Philadelphia: P. Blakiston's Son & Co., 1931. Clark, W. M.: Determination of Hydrogen Ions, Baltimore: Williams & Watkins Company, 1928.

20. Sansum, W. B.: Normal Diet, St. Louis: C. V. Mosby Co., 1930, p. 122.

21. Hess, A. F.: Rickets, Osteomalacia and Tetany, Philadelphia: Lea & Febiger, 1929, p. 133.

22. Jones, Martha R.: Studies in Inorganic Salt Metabolism, Proc. Soc. Exper. Biol. & Med., 21:199, 1924.

23. Hodgson, A.: Vitamin Deficiency and Factors in Metabolism, Relative to Development of Rickets, Lancet, 2:945 (Nov. 5) 1921.

24. György, P., and Stepp: Avitaminosen, Berlin: Julius Springer, 1929.

25. Blatherwick, N. R., and Long, M. L.: Studies in Urinary Acidity. 1. Some Effects of Drinking Large Amounts of Orange Juice and Sour Milk, J. Biol. Chem., 53:103 (July) 1922.

26. Bunting, R. W., et al.: Problem of Dental Caries, Am. J. Dis. Child., 40:536, (Sept.) 1930. Koehne, Martha: Present-Day Theories of Cause of Dental Caries, J. Am. Diet Ass'n, 7:335 (March) 1932. Bunting, R. W.; Jay, Philip; Hard, Dorothy G.: Report of Successful Control of Dental Caries in Three Public Institutions, J.A.D.A., 18:672 (April) 1931.

27. Hanke, M. T.: Relation of Diet to Caries and Other Dental Disorders, J.A.D.A., 16:2263 (Dec.) 1929; Relation of Diet to General Health and Dental Caries, ibid., 17:957 (June) 1930. Rôle of Diet in Cause, Prevention and Cure of Dental Diseases, J. Nutrition, 3:433 (Jan.) 1931.

this fruit. Boyd and Drain,[28] of Iowa City, have actually produced enamelization inside the pockets of carious teeth by diet. I had the privilege recently of examining with a sharp dental pick the teeth of two of their children in whom this epoch-making cure had occurred. One child with all cavities healed was a diabetic who had been in the institution for three years. The other child was reasonably near normal, but had been on a similar diet for three months. One cavity in this second child's mouth was well enameled and another partly so. The bottom and sides of the healed cavities were as hard as stone. From the menus shown me by the dietitian, the foods consumed were largely from the base-forming groups. The diets provided 1 gm. of protein per pound of body weight and a fatty acid to glucose ratio of 1:1. Particular emphasis was placed on milk, vegetables and fruits.[28] Although the biologic effects of these diets as shown by the urinary p_H had not been determined, such diets would presumably produce a neutral or slightly alkaline urine.

The three groups of workers at Ann Arbor, Chicago and Iowa City who have prevented or cured caries give their diets the credit. The common factor in these diets is the preponderance of base-forming foods. Corroborative experimental evidence is furnished by Howe,[29] who found that monkeys fed on a milk, vegetable and fruit diet have excellent teeth. This corresponds also with the conclusions (contradicting to a certain extent the former statement of Jones[22] quoted above,) drawn by Jones, Larsen and Pritchard[30] after a careful study of Hawaiian children: "Fruits and vegetables were invariably found to be associated with sound tooth structure or arrested decay when they constituted a sufficiently large proportion of the diet to render the ash alkaline in reaction," and again, "All the evidence available indicates that dental decay is primarily a systemic disorder, an imbalance perhaps in the tissue fluids and oral secretions in the direction of a decrease in basic ions either relative or absolute."

28. Boyd, H. D.; Drain, C. L., and Nelson, M. V.: Dietary Control of Dental Caries, Am. J. Dis. Child., 38:731 (Oct.) 1929; Dietary Control of Dental Caries, J.A.D.A., 17:738 (April) 1930; Control of Dental Caries, Pub. Health Bull., March, 1931.

29. Howe, P. R.: Investigation of Dental Caries, J.A.D.A., 14:1864 (Oct.) 1927.

SALT-POOR DIETS

Another factor in modern diets which has come to occupy a place of importance in nutritional study is common table salt, sodium chloride. Before the days of ice and artificial refrigeration, salt was needed for preservation of food. This use of it has accustomed civilized peoples to its taste so that many demand a considerable amount.

Is it not possible that civilized people have teeth inferior to those of primitive folk in part because of this excessive use of salt? Janet Miller,[31] in "Jungles Preferred," mentions no tooth troubles in darkest Africa except those due to the wearing down of the crowns in the very old people, and these people had had only the salt natural to foodstuffs. Their diet consisted mainly of unsalted fruits and vegetables.

It seems probable that meat from animals fed large amounts of salt would taste different from that of those given a salt-poor diet. Ripley states that Fiji Island cannibals had an aversion to the white man because his flesh was too salty. These same cannibals are reported to have had

30. Jones, Martha R.; Larsen, N. P., and Pritchard, G. P.: Dental Disease in Hawaii, III. Factors in Dental Decay in Preschool Children, D. Cosmos, 72:685 (July) 1930.

31. Miller, Janet: Jungles Preferred, Boston: Houghton Mifflin Company, 1931.

better teeth than their white victims. Weld, in his book, "Manners and Customs in Philadelphia," stated, in 1795, that American born men and women had decayed teeth at an early age. He thought that this was due to the very general use of salted provisions. He eliminated climate as a causative agent since the Negroes and Indians had sound teeth. McCollum[6] quotes Catlin as saying that the skulls of the Sioux and Iowa Indians in the Mississippi valley had teeth as regular as the keys of a piano, and that the Indians were astounded at the derangement and absence of teeth among white people.

The very prairies that once produced people with comparatively perfect teeth now produce individuals with inferior teeth. Since primitive peoples in the tropic and temperate zones have good teeth, let us consider the situation in the Arctics. Eskimos living largely on fatty fish and blubber, neutral-ash foods, and having little vitamin C or sunlight, are reported to have excellent teeth until they come in contact with our salt-preserved foods.

The salt hypothesis, therefore, it seems to me, deserves serious consideration. We have been taught that the use of sodium chloride is necessary. We use it not only as a preservative, but also as a condiment, and our appetites have been cultivated to demand a certain amount. Unlike most of the salts in our food, sodium chloride is free from oxygen. In the ash analysis of the human body, we found equal amounts of the metal sodium and the acid-former chlorine. No organic compounds containing chlorine are known to exist in the body. The alkaline sodium ion can be exchanged for other bases and is useful as a buffer, but chlorine is of no known value as a body builder. Its use in the formation of hydrochloric acid in the stomach is essential, but here it is not lost to the body because it is changed into sodium chloride in the intestines and reabsorbed again. Probably the chief function of sodium chloride in the body is to render proteins soluble. But for the salt in the blood (0.8 per cent), liquidity and osmosis would be interfered with. Hast[32] reminds us that the life and activity of the body depend on certain physiochemical qualities of the fluids of the body: fixed osmotic pressure, and fixed hydrogen ion concentration. He claims that to get a diet too low in salt would require the extraction of salt from natural foodstuffs.

The amount of salt needed daily by a normal adult is estimated by Strauss[33] as from 1 to 1.5 gm. Most of us eat from 10 to 25 gm. a day. Each gram of salt retained in the body holds in the tissues 70 gm. of water. A man weighing 150 pounds may retain 1 gallon of water and not show edema. This excess amount of liquid dilutes and hence alters all the fluids of the body. The lymphatic fluids of the teeth must be affected by this dilution and thereby have their nutritional qualities reduced. This excess fluid likewise increases exudation from mucous membranes. Ullman[17] has shown that chronic sinusitis is often cured by giving a salt-poor diet, as it produces a dehydration of the membranes, which decreases infection.

Urbach,[34] reviewing the work done on salt metabolism of the skin, shows that the skin contains approximately 30 per cent of the total chlorides in the body and that decreasing the salt in the diet will cure certain conditions such as keloids and

32. Hast, H. H.: Chloride Metabolism, J. Lab. & Clin. Med., 5:713, 1920.

33. Strauss, H.: Zur Frage der Diatbehandlung der Lungentuberculose, Med. Klinik, 25:1383 (Sept. 6) 1929.

34. Urbach, E.: Skin Diseases and Nutrition, Vienna: Wilheim Maudrich, 1932, p. 16.

lupus. Recent work at the Mayo Clinic[35] corroborates the findings of others in proving the value of salt-poor diets in cutaneous and osseous tuberculosis. Mayer and Kugelmass,[36] using this diet in a group of cases of severe tuberculosis of the lungs, report improvement after other treatment had failed.

SODIUM VERSUS CALCIUM

An important point in the relation of sodium chloride to rickets and caries is its effect on calcium metabolism. The biochemists tell us that the cation sodium will expel the cation calcium. Certainly, a solution that is saturated with sodium can absorb very little calcium. Kugelmass[37] emphasizes the physiochemical fact that living cells already saturated with electropositive sodium ions find the utilization of calcium difficult. Von Noorden[38] has shown that a person can retain approximately 2 gm. of calcium oxide a week for a certain period on a base-forming and salt-poor diet. If, therefore, we reduce sodium in the diet, the absorption and retention of calcium is more easily increased.

As far as I am able to learn, this idea of the diluting of the nutrient fluids in the body caused by an excess of sodium chloride and the decalcifying effect of sodium chloride has not heretofore been applied to the teeth. Additional clinical and experimental evidence is obtainable without undue difficulty and, it is hoped, will soon be available.

COMMENT

The effects of mineral deficiency observable in the bones of children, such as in knock-knees, flat feet, round shoulders and sunken chests, become evident in the teeth from four to six years later. One great difficulty in the study of the relation of diet to dentition in human beings is the long period which must elapse between the cause and the visible result. Being a matter of years, it is difficult to trace a defect back to its origin or to follow a case until the result is demonstrable.

After infancy, the most strategic period of life from the standpoint of mineral requirements is adolescence. At this age, we find rickets developing even in children that have previously escaped the disease. Dentists well know the prevalence of caries during the teen age or later adolescence.

The amounts of calcium and phosphorus needed by a growing child are obtained by the consumption of a varied diet containing a quart of milk daily. With young children, a quart of milk may make the protein percentage too high or cause anorexia[39] or anemia.[40] Also, an

35. Dörffel, J.: Effect of Diet Low in Salt in Cases of Tuberculosis of Skin, Proc. Staff Meetings Mayo Clinic, 7:73 (Feb. 10) 1932.

36. Mayer, E., and Kugelmass, I. N.: Basic (Vitamin) Feeding in Tuberculosis, J.A.M.A., 93:1856 (Dec. 14) 1929.

37. Kugelmass, I. N.: Buffer Mechanism for Calcium Concentration and Determination of Calcium Buffer Values, J. Biol. Chem., 60:237 (June) 1924; Feeding in Infancy and Childhood, Philadelphia: J. P. Lippincott Company, 1930.

38. Von Noorden, Carl: Alte und neuzeitliche Ernahrungsfragen unter mitberucksichtigung wirtschaftlicher Gesichtpunke, Berlin: Julius Springer, 1931; Ueber Zickzakost, Therap. d. Gegenw., 72:11 (Jan.) 1931.

39. Schlutz, F. D.: Problem of Chronic Anorexia in Childhood, J.A.M.A., 94:73 (Jan. 11) 1930.

40. Daniels, Amy L., and Hutton, M. N.: Mineral Deficiencies of Milk as shown by Growth and Fertility of White Rats. J. Biol. Chem., 63:143 (Feb.) 1925. Orten, J. M.; Underhill, F. A., and Lewis, R. C.: Study of Certain Metals in Prevention of Nutritional Anemia in Rat, J. Biol. Chem., 96:1-9 (April) 1932. Beard, H. H., and Myers, V. C.: Studies in Nutritional Anemia of Rat, 94:71 (Nov.) 1931. Hart, E. B., et al.: Nutritional Anemia on Whole Milk Diets and Utilization of Inorganic Iron in Hemoglobin Building, J. Biol. Chem., 65:67 (Aug.) 1925.

individual may be allergic to milk or for some other physiologic reason be unable to assimilate a quart of milk daily. If so, from what other sources may an equivalent amount of these basic minerals be obtained? For the solution of this and similar problems, recent books[41] and the suggestions of well-trained dietitians are indispensable.

The discovery of vitamins altered the previously held ideas of mineral metabolism and will continue to modify conceptions of the subject until all the vitamin factors are definitely known and optimum quantitative requirements determined. For instance, with an adequate intake of vitamin D or with heliotherapy, the calcium intake can safely be lower than when the reverse condition exists. The discovery of vitamins has also stimulated nutritional research and educational propaganda along food lines. The commercial companies have assisted greatly in popularizing foods containing vitamins. Since the vitamins are found mostly in alkaline foods, diets have been altered in a single decade from highly acidic ones to a more basic type. Diets in general now contain more milk, vegetables and fruits than formerly. By this means, the mineral content has been so changed as to make the positive electrolytes, the basic elements, more prominent. Is it not now time to give the minerals the credit they deserve and broadcast the importance of base-forming elements in the diet? Fortunately, a monthly examination of a twenty-four-hour urine specimen (using thymol or other preservative) for its hydrogen-ion concentration and for total chlorides determines definitely whether the diet is permitting bodily functions to be performed economically and whether the mineral metabolism is reasonably near normal.

It is evident that the factors in forming and maintaining sound teeth are many and varied. Animal experimentation and individual human cases can only point the way. Conclusions need corroboration through observation of a large number of cases.

SUMMARY AND CONCLUSIONS

1. Using the ash of the human body and of its various organs as a basis for the study of inorganic mineral requirements, one finds a major group of elements containing only calcium and phosphorus; a minor group containing potassium, sulphur, sodium, chlorine, magnesium, iron and iodine, and a "trace" group of immense importance, including fluorine, silicon, copper, aluminum, zinc and manganese.

2. The metabolism of minerals is dependent on the intake of foods in balanced proportions and the maintenance of a state of slight alkalinity (p_H 7.4) in the tissues and fluids of the body.

3. The classification of foods according to their ash content into acid, neutral and alkaline assists in the arrangement of menus so as to have the least possible nutritional friction during utilization in the body.

4. Evaluating foods according to their biologic effect is easily possible because the nonrespirable ash is excreted largely

41. Footnotes 2, 7, 8, 20 and 34. Blum, Sanford: Practical Dietetics for Adults and Children, Ed. 4, Philadelphia: F. A. Davis Company, 1931. Cannon, W. B.: Wisdom of Body, New York: W. W. Norton Company, Inc., 1932. Gauss, Harry: Clinical Dietetics, St. Louis: C. V. Mosby Company, 1931. Harrop, G. A.: Diet in Disease, Philadelphia: Blakiston's Son and Company, 1930. Marriott, W. McK.: Infant Nutrition, St. Louis: C. V. Mosby Company, 1930. McLester, J. S.: Nutrition and Diet in Health and Disease, Philadelphia: W. B. Saunders Company, 1927.

by the kidneys. Determination of the hydrogen-ion concentration of a twenty-four-hour urine specimen proves the correctness of a diet.

5. Base-forming diets containing a preponderance of fruits and vegetables prevent the development of odontoclasia and tooth decay, and enable a carious pocket to reenamel itself. Among vegetables, potatoes deserve special consideration.

6. Historical, chemical and clinical data support the hypothesis that salt-poor diets diminish focal infection and dental caries and improve the assimilation of calcium.

7. Available evidence indicates that the etiologic factors involved in tooth decay are more systemic than local and that an adequate diet will prevent, check or cure caries.

8. Close cooperation between the research laboratory and the clinician and between the dietitian, the doctor and the dentist is necessary to solve the nutritional problem of building decay-resistant teeth.

MILD PRESSURE AND GREAT FORCE

By J. E. RICHMOND, D.D.S., Eugene, Ore.

FOR more than fifty years, men have been writing in ever increasing abundance and, we trust, with an increasingly scientific attitude, concerning the movement of teeth.

As early as 1888, Levitt E. Custer wrote in the *Ohio Journal of Dental Science*: "Resorption, as a physiologic process, takes place under the agency of a class of cells which may be understood to have a retrograde function . . . Such cells . . . have been termed . . . osteoclasts. . . . Osteoclasts are also stimulated by pressure, and upon the latter do we depend for change of shape in the alveolar process for correcting irregularities."[1]

Before and since that time, appliances of various forms and using different types of wire have been developed, every one with the single purpose of exerting pressure on teeth to move them.

Dewey's "Practical Orthodontia"[2] says, "It is true that teeth have been moved and can be moved by the application of a great amount of force and such movement occurs too often, but the movement is not physiologic and those who use such methods are not practicing orthodontia. They are only moving teeth and causing much suffering to their patients."

That excessive pressure to teeth will cause undesirable results was known at least as early as 1873, yet up to the present there has been no method of determining exact amounts of pressure and but two pressures are mentioned in orthodontic literature: mild pressure and great force. There seems to be no unit of force which is in common use by the profession, nor any means in the hands of the orthodontist for gaging in terms of a given unit

1. Weinberger, B. W.: Orthodontics, St. Louis: C. V. Mosby Company, Vol. 2, 1926, p. 745.

2. Dewey, Martin: Practical Orthodontia, Ed. 3, St. Louis: C. V. Mosby Company, 1917, p. 157.

the force that he puts on teeth even though he knows what force is most desirable.

I know of but two exceptions to the foregoing. J. E. Johnson says, "To my surprise, I found the average pressure on the tooth ranges from 2 to 10 ounces, depending on the irregularity of the teeth." He also says that he had discovered that an 18-gage arch wire, ligated to the teeth, often exerts from 2 to 4 pounds of pressure. Several years ago, I read a paper by someone who recommended the use of not more than 3 ounces of pressure on a tooth. With these exceptions, I have found no definite statements of the number of ounces of pressure that is recommended for moving teeth though I have searched the textbooks that violent pressure causes the active bone cells to completely leave the field, thus making physiologic movement impossible.

To the extent of proving the desirability of mild force for moving teeth, the experiment seems to have been successful, but the value to the practicing orthodontist would be much greater if Oppenheim had known and recorded just how many ounces of pressure he had used as a mild force and whether his violent force was 1 pound or 6. He may have considered 1 pound a mild force or he may have found that 1 ounce was sufficient to move teeth. He probably had no means of weighing the pressure he used and so leaves us very much in the dark on an extremely important part of

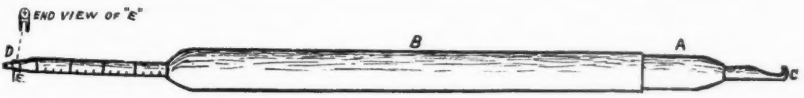

Fig. I

Fig. 1.—Orthodontic pressure and tension gage.

of Angle, Dewey and McCoy and the two volumes on "Orthodontics" by Weinberger; as well as a large number of magazines devoted to orthodontia and dentistry.

In McCoy's "Applied Orthodontia,"[3] there is a résumé of "Oppenheim's experimental evidence" which tells of orthodontic appliances being put on the teeth of several young baboons. It says, in part, "In each instance, after applying pressure of different degrees . . . the animal was killed and histological preparations were made of the parts subjected to pressure as well as the parts used as control. . . . " The experiment seemed to prove that mild pressure stimulates the action of osteoclasts and osteoblasts, but the experiment. Even had he told us the exact pressure used, we should still need some definite means of applying the pressure determined to be most desirable.

Dr. Ketcham published quite an extended article[4] on the resorption of roots in orthodontic cases. He had tabulated the cases showing the type of appliance that had been used in every case, but there was nothing to indicate that any check had been kept on the *amount* of pressure that had been used. He came to the conclusion that the most resorption occurred when these types of appliances capable of producing root movement, especially Angle appliances, were used. The tests, of which I will speak later, show that few men are able to "guess" within 75

3. McCoy, J. D.: Applied Orthodontia, Philadelphia: Lea & Febiger, 1922, p. 157.

4. Ketcham, A. H.: Internat. J. Orthodon., 15:310 (April) 1929.

per cent of the amount of pressure that is exerted on a given attachment and that the amount of torque exerted by a ribbon arch or a pin and tube attachment is the most deceiving. It seems reasonable to suppose, therefore, that had these pressures been measured and a record kept, the resulting resorption could have been laid more properly to disturbed circulation caused by two great forces being exerted at the apex rather than to the appliance *per se*.

Pressure on teeth has a definite parallel with the drug strychnine; in small doses, both are stimulant; in large doses, both will produce disastrous results. Suppose

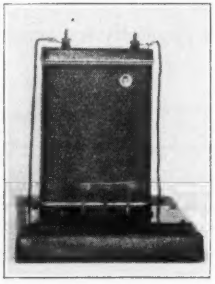

Fig. 2.—Testing apparatus to determine force exerted by different wires when sprung definite distances.

that the Materia Medica treated strychnine as orthodontic literature treats pressure; i. e., suppose the dosage were given thus: A very minute dose is a good stimulant, but too large a dose will cause death, and then suppose that scales had not yet been invented, the physician being compelled to estimate each dose by dipping the point of his knife into a box of strychnine. Occasionally, he might give too small a dose to do any good, and it is presumed that he might occasionally "guess" just the right amount, but beyond a doubt, taken by and large, the medical profession would very often be too liberal and the patient would cease to exist.

We would think that such procedure was absolutely unscientific, and so it is; yet this exactly parallels the way that orthodontic pressure has been applied; without a definite dosage or any scales to assist in establishing one.

Dr. Johnson says that since the roentgenograms of the teeth that he treated revealed no absorpion of the roots or process, one might assume that from 2 to 10 ounces is not an excessive pressure. Either 2 or 10 ounces, some amount in between

Fig. 3.—Testing apparatus in use.

or less than the 2 or more than the 10 is the most *desirable* pressure; that is, the one that will be the best to stimulate the activity of the bone cells without causing inflammation. Of course, this will vary some with the case. As the veterinary Materia Medica says "The dose for a dog is one-half that for a man (but consider the size of the dog)."

The realization that orthodontic pressure should be measured has been growing on me for some time and the picture of Dr. Johnson's pressure gage in the article mentioned above caused me to

write him immediately to find out where one might be obtained. He replied that it was his own personal product and had been made by removing the pan from an ordinary postal scale and shaping the standard so that it could be applied to the wire to be tested. Though this disappointed me, it at the same time stimulated me to contrive a pressure gage for myself. With my ideas partly formed, I went to an acquaintance who is equipped to manufacture instruments and together we devised an instrument that looks like an automatic gold plugger and will weigh forces either by pressure or by traction. The first plan was to have an instrument

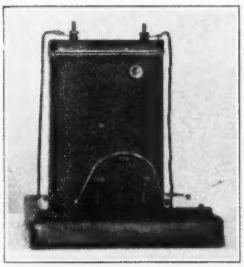

Fig. 4.—Apparatus used to test arch wire force when it is supported only by buccal tubes in molars.

that would weigh forces up to 16 ounces, calibrated in ounces, but as it was later decided that a more delicate adjustment would be more desirable in many places, one was made to weigh up to 4 ounces graduated in one quarter ounces. At present, the use of both instruments is found desirable.

In practice, the point (Fig. 1) *D* is placed on the arch wire or finger spring in its rest position. The instrument is then used to press the wire to its working position. The number of ounces of pressure used will be the reaction on the tooth when the wire is fastened to position whether by ribbon arch bracket, open tube, pin and tube, ligature or otherwise. When a root-moving attachment is used, it is possible to so adjust it that the greater part of the pressure is exerted on the apex of the tooth, and it is surprisingly easy to develop pressures at this point that would so crush the periapical tissues as to make certain changes in circulation sufficient to account for the root resorption that is so often reported in this class of movement.

E, Figure 1, shows an attachment suggested by Dr. Johnson for testing the pressure on coil springs. The slot is slipped over the arch wire carrying the coil spring, the arms resting against the end of the coil on both sides of the arch wire.

In order to make laboratory tests of the reaction of various wires when sprung definite distances, the apparatus shown in Figure 2 was constructed. It consists of a plate carrying three screws in a row. The outside screws are attached to one side of a dry battery and the center screw is attached to the other in such a manner that when the contact is made between the end screw and the center one, the lamp is lighted. By laying a wire, to be tested, from one outside screw to the other, we have an example of an arch wire at rest. When the center screw is turned so as to raise it until it just touches the wire and lights the lamp, we have a definite starting point. Each complete revolution of the center screw will lower it one-fiftieth inch (there being fifty threads to the inch). Therefore, the distance that it will be necessary to spring the wire (to what would represent its working position) can be accurately gaged to one two-hundredth inch, if desired.

Figure 3 shows the gage at work with the wire sprung down to make contact with the center screw and light the lamp.

The distance between the outside screws is 21 mm., which represents the distance between the most prominent point on a central incisor on one side of the mouth and a lateral incisor on the other. In other words, it will give the same amount of spring to the wire as though it rested on a central and lateral incisor with the pressure exerted to move the other central incisor labially.

Figure 4 shows an arch fitted into tubes that are the same distance apart as tubes on molar bands in the mouth. With this adaptation, the tension on an arch, which does not rest on any other teeth, can be measured.

The hook marked *C*, Figure 1, is for testing elastic ligatures or finger springs that are exerting pressure rather than pull.

The accompanying table gives the pressures needed to deflect different gages of wire for various distances. "The distance between rest points" approximates various points on which the arch wire might find a fulcrum and is the "length of the spring base" used in each test.

As the standard tension gage is made to weight up to 16 ounces only, it was necessary to construct one with a heavier spring to weigh reactions over 1 pound. The tension on the old style German silver arch when sprung one-fiftieth inch (or 0.5 mm.) was probably 5½ pounds. Certainly this would be considered, by the most liberal, as an excessive pressure, yet I have been informed by recent graduates of class A dental colleges that, in their class lectures, they were never advised of the importance of keeping orthodontic pressure within physiologic limits nor of the dangers of excessive pressure. They said that a German silver arch was merely adjusted in the buccal tubes of the molar bands and they were instructed to ligate it to the teeth to be moved; and that they now realized, from the tests shown them, that the pressures applied must have been several pounds.

It will take the records of several hundred cases to get a definition of "a mild pressure" (within physiologic limits) and to discover at what point an excessive pressure is reached.

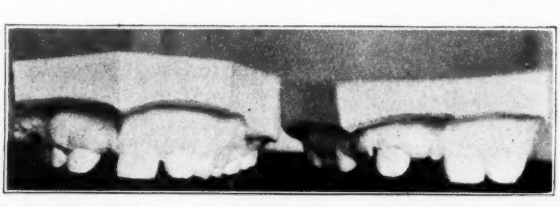

Fig. 5.—Case before and after force of 2 ounces was applied for twenty-three days. The space was increased 3.2 mm.

Figure 5 shows a very simple case of tooth movement under a measured force of two ounces. These are the models of the teeth of a boy, aged 8 years, who had lost the right central incisor, and the other central incisor and the lateral incisor had drifted together until the space measured but 6 mm. (the other central incisor measured 9.2 mm.). The central and lateral incisors were banded with bands fitted with ribbon arch brackets. A simple loop of 24-gage gold-platinum wire was fitted to these brackets, and it was found that when the wire was adjusted so that it would be sprung about 1.5 mm. to be placed in the bracket, it would exert 2 ounces of pressure. The appliance was put on September 8 and was adjusted

September 15 and 22, each time with 2 ounces of pressure. September 29, the boy came in for an adjustment, but as the space was found to be sufficient for the replacement of the lost incisor, no adjustment was made. October 1 (just twenty-three days after the appliance was placed), the appliance was removed and the impression for the second model made, and the boy was sent to his dentist to have an impression taken to replace the lost tooth. Then the appliance was replaced to hold the teeth in position until the dentist's work should be completed.

PRESSURE REQUIRED TO DEFLECT GAGES VARIOUS DISTANCES

Distance Between Rest Points Mm.	Gage of Wire	Amount of Deflection Inch	Reaction in Ounces
21	24	1–50	3¾
21	24	1–25	7½
21	21	1–50	13
21	21	1–25	26
21	0.030	1–100	10
21	0.030	1–50	20
21	0.030	1–25	40
21	0.036(19)	1–100	24
21	0.036(19)	1–50	40
21	0.036(19)	1–25	64 (4 lb.)
Standard German Silver Arch	17	1–100	44 (2¾ lb.)
	17	1–50	(Scale weighed up to only 4 pounds)

As nearly as could be measured, the lateral incisor moved 2 mm. and the central, 1.2 mm.

The teeth never became in the least sore and seemed to be as solid after this rapid movement as before; which would lead to the conclusion that it is not the rapidity of movement that causes looseness in teeth, but rather the fact that they are moved by an inflammatory reaction rather than by cell activity. When one considers that a broken bone will often be completely healed in three weeks, it seems perfectly reasonable to suppose that bone reconstruction might be complete after moving a tooth a distance of 2 mm. in the same length of time, if cell activity is normal.

Of course, no one case establishes any facts, but if orthodontists generally can be induced to use definite weighed pressures and tabulate them with the age of the patient and results, more practical information as to what constitutes the most desirable pressure for the movement of teeth may be developed in a few months than all of the experiments on animals have so far produced.

RECAPITULATION

Though past experiments and observation have established the fact that only mild pressure will move teeth by physiologic reaction, nothing seems to have been done to establish (in ounces) just what may be the limits of "mild pressure."

Even when the most desirable pressure is established, there has been no instrument put in the hands of the individual orthodontist to enable him to use that pressure accurately.

Teeth will move faster and with less discomfort to the patient and be more quickly stabilized in their new position if the force used is within physiologic limits and these limits could be quickly ascertained (in ounces) if a large number of orthodontists would tabulate and report cases showing the pressures used and results obtained.

Excessive pressures are not uncommon, because of the inability of the operator to weigh his pressure and the deceptive ease with which excessive pressures are produced.

In some dental colleges (and I fear in many), little if anything is taught the students as to the evil results of great

pressure, and men are so naturally mechanically minded that desire for action automatically calls for added pressure unless they have been taught the facts of tooth movement.

As mild a pressure as desired *might* be applied by any type of an arch but the more rigid the wire, the less it will be deflected to produce a certain pressure, and the movement in the tooth cannot be greater than the deflection of the wire; therefore, the lighter wires will produce greater movement than will the heavier wires without having excessive pressure at the start.

Root resorption has been laid to the types of appliance used, but there seems to be more reason to think that it is due to too great force being applied at the apex, which results in a bruising of the periapical tissues and thus in disturbance of the normal circulation.

THE GOLD FOIL STUDY CLUB*

By GEORGE N. HOLLENBACK, D.D.S., Los Angeles, Calif.

THE study club movement has played a rôle in the advancement of the science and practice of dentistry which is perhaps secondary to that of no other. Many clubs have been organized throughout the country for the purpose of conducting study and research in all the various factors of dental practice.

It would seem evident at this time that those clubs which have devoted their efforts almost exclusively to the problems of operative dentistry, and more particularly to gold foil, have been longer lived and perhaps have more to their credit in the way of improvement of the knowledge and technical skill of their members than have clubs devoted to other phases of dental practice. It seems that the perfection of an operator in gold foil technic not only gives him the knowledge whereby other phases of operative dentistry can be more successfully handled but also much better fits him to cope successfully with the many problems constantly arising in dental practice outside of the operative field.

Gold foil tends to develop in the operator a more idealistic point of view because it permits him to do his utmost in the expression of his artistic ability and skill. It is perhaps the only procedure in dentistry whereby a perfect result may be produced, though the road to perfection is long and arduous and few there are who can hope to achieve the ultimate goal. It is the continual striving for this goal that develops within the operator the knowledge, manual dexterity and artistic sense which go to make up the personality of the successful practitioner.

A very large percentage of the men who, in the past, have made dental history and those who are making it today have been and are skilled foil operators. There are few, if any, radicals in their ranks. In fact, they perhaps constitute the most safe, sane and conservative group in the profession. Their very training is such that while they are progressive and wide awake, they are always conservative in

*Many foil clubs not mentioned have been organized and successfully operated, but space does not permit their inclusion in this brief history.

overemphasizing the new and untried. Therefore, in this hectic day of ever-changing technics and the commercial exploitation of innovations of doubtful merit, this group may well serve as a balance wheel and criterion for the entire profession.

The first gold foil study club was organized by the late E. K. Wedelstaedt, of St. Paul, May 20, 1898. This club was known as the G. V. Black Study Club. During several years previous to this, Dr. Wedelstaedt had been closely associated with Dr. Black, and it was the inspiration which this association gave him which proved the guiding spirit of the rest of his life. Appreciating the great necessity of a more general application of Black's principles, he, with the aid of A. C. Searle and R. B. Wilson, organized this club. The object was the improvement of its members through the medium of instruction in gold foil technic. This movement met with the enthusiastic and whole-hearted support, of the profession and its direct and indirect effect in raising the standards of dental practice can hardly be overestimated.

The club, which was incorporated in 1902, active membership being limited to twenty-five, with no restriction on the number of associate members, has operated continuously since its organization and, during this time, approximately 200 operators have been trained. It has never deviated from the ideals of its founder, practically every meeting being devoted to gold foil technic. The fact that this club has met six or seven times a year for more than thirty-four years indicates a most commendable steadfastness of purpose and devotion to an ideal. Its members have displayed excellent judgment and balance in adhering to a technic which has stood the test of time better than any other.

It has been this very devotion to an ideal which has kept the manipulation of gold foil from becoming largely a lost art. The trend back to gold foil which is so manifest in dental circles today and the good which will be accomplished thereby is directly attributable to the teaching of these groups. It was a natural consequence of the success of the Black Study Club that similar groups were organized in other parts of the country. The history of some of these clubs will be very briefly given.

C. E. Woodbury, of Council Bluffs, Iowa, one of the early associate members of the Black club, became so thoroughly impressed with its work that, in May, 1906, he organized a similar club in his city, its objectives and ideals being the same as those of the Black club. This club was founded with twenty charter members and it also made provision for associate members. It has been in continuous operation since it was founded, meeting two or more times each year. It, like the Black club, has been a great factor in raising the standards of dental practice in the middle west and it has to its credit the development of a number of the finest operative dentists in America today.

Another organization contemporary with the Woodbury club was the E. K. Wedelstaedt club, of northern Iowa, which, during its comparatively brief life of five or six years, developed a number of operators who today stand high in the ranks of the profession.

A few years later, H. E. Latcham, of Jefferson, Iowa, organized in central Iowa, a club called the J. M. Prime Study Club, with a membership of ten men. This name was selected in appreciation of the work of James M. Prime, of Omaha, who has done so much in spreading the gospel of gold foil and the teach-

ing of gold foil technic. This club survived for several years, and it also did much to improve the technic of its members and to raise the standards of dental practice in that locality.

(The credit for applying Black's principles in a more conservative manner without sacrificing the spirit of his teaching should go to Dr. Prime to a greater extent than to any other individual. As a conserver of dental tissue, he occupies the first rank and his teaching along this line has had a most profound effect throughout the entire country.)

Another club, known as the Prime Research and Gold Foil Club, was organized about three years ago in Omaha, Nebr. This club has twelve members who devote two half days a month to its activities. I have had occasion to examine the work of some of its members and have found that, even in this brief period, remarkable progress had been made.

The foregoing constitutes a somewhat brief history of the organization of gold foil clubs in the middle west, where this movement had its inception. An attempt will be made to give in a somewhat more detailed way the history of similar movements in the Pacific Northwest. Owing to close personal contact with the leaders in this movement over a number of years, I feel that I am in a position to speak with considerable authority of the results that are being achieved in this particular locality also, to present in a somewhat detailed manner the plan on which these clubs have been organized and so successfully conducted and to further point out the fact that other localities which may become interested in this movement will do well to make a careful study of this plan which, from the results achieved, would seem to be well nigh perfect. It is obvious that, for this or any similar plan to be successfully followed, it must be put in practice by one who has true qualities of leadership. The fact that Seattle has a dentist who possesses to a marked degree the qualities of leadership and the willingness to labor indefatigably as a teacher and clinician of gold foil technic has been one of the outstanding reasons for the great success of the movement in this particular locality. W. I. Ferrier, in my opinion, from the standpoint of ability and achievement ranks as one of the greatest operators of all time.

The study club movement had its inception in Seattle about twenty years ago, when Drs. Searl and Wedelstaedt were induced to come there and give instruction to twelve men, among them Dr. Ferrier, who was then located in Burlington, a small town about 80 miles from Seattle. This group formed a foil study club known as the Seattle Dental Study Club, and, during the next eight years, meetings were held each month, the entire time being spent in actual operations in the mouth. This club pioneered the gold foil movement in the Pacific Northwest and was instrumental in organizing similar clubs in this section. Much credit is due the men that then comprised its membership for their efforts. However, their numbers were reduced to such a point, through lack of leadership perhaps, for every group of this kind must have a leader if it is to achieve the ultimate, that it was necessary to form an entire new group of younger men to carry on the work and preserve the identity of the organization.

About twelve years ago, Dr. Ferrier, who had in the meantime located in Seattle, was asked to assume the directorship of this group and give its members a week's intensive instruction, the work to be continued indefinitely through the medium of regular meetings throughout the year. The members of this group,

believing that all should have a proper feeling of responsibility and interest in their organization, decided that their director should be paid for the time spent in directing their activities. This arrangement was not brought about at the suggestion of the director but was done entirely of their own violition and that this arrangement has been a success is well attested by the outstanding results achieved during the ensuing years. It is a strange characteristic of human nature that, as a rule, anything, be it ever so good, is not appreciated if it can be secured without cost to its recipients. Conversely, something of no greater merit, but whose acquisition requires something in the nature of financial outlay, is usually enthusiastically received and appreciated.

In passing, it might not be inapropos to cite the many study clubs throughout the country that have failed to operate successfully for long, although the members have certainly been actuated by altruistic motives and teachers and directors, likewise actuated, have given unsparingly of their time and effort in their respective capacities, asking for no renumeration whatsoever and, in many cases, even going so far as to help to defray the expenses of the organization themselves. This is in no way an argument in behalf of the commercial dental instructor (whose activities are certainly seldom actuated by altruism), but rather a statement of a condition which the experience of many persons has shown conclusively to exist.

In behalf of the paid director of a study club, much can be said, and it is a fact that few will dispute that many excellent teachers are not financially able to give of their time and effort without recompense and, if some arrangement is not made for payment for the service rendered, either their services will not be available or the personal sacrifice will be altogether too great. Another and very valid argument in behalf of the paid director is that this one provision automatically eliminates all but earnest workers from the membership of a club. It can readily be appreciated that if membership in a club entails little financial outlay, many who are not worthy will be attracted to its ranks, usually by motives which, to say the least, are not laudable.

The success of this small group attracted a great deal of attention, and its members, entirely without solicitation on their part, were in constant demand as clinicians before local and various state organizations and the Pacific Coast Dental Conference. Many others wished to become members of this club, but this was impossible as it was believed that a membership of more than twelve would be unwieldy. This was a serious problem as many who desired membership were worthy and actuated by the best motives and were certainly entitled to every consideration. This matter and its solution can best be expressed in the words of Dr. Ferrier, in a letter received recently, part of which is as follows.

> How could earnest men who wished this type of work be taken care of? From experience I knew that a group of twelve is the best working number. But it was not right nor in keeping with traditional professional conduct to deny men who earnestly desired knowledge the opportunity to obtain it. So a plan was devised whereby groups of twelve could be formed and if they complied with the requirement they could start as we did under our instruction, guidance and help. A two weeks' course was given by me with the aid of the members of the original club. The proportion of one instructor to every six men was our requirement. Another was that they must continue as a group and function as we do with a meeting each month and be instructed by one of the men who helped me in the course. A fee was charged for instruction to such an amount that the instructors could give their time without too much

loss. . . . Now had we not made these requirements, many who were not sufficiently interested would have sought our services and the whole movement would have been a total failure for the following reasons. A small financial interest is necessary to a going organization; gold foil is not for everyone nor is the finer technic in other branches of dentistry. Those willing to comply with the requirements are usually of the type that will make good. . . . Six clubs are functioning under this plan with a membership of seventy-six men. We have provided six instructors with myself from the Seattle Study Club to carry this on.

That this work is being well carried on was amply attested by the operative clinic given during the Pacific Coast Dental Conference held in Seattle last July. This clinic was pronounced by many who witnessed it as the most impressive thing they had ever seen. Of a total of thirty-three operators, all but two or three were members of study clubs in Seattle and Portland, and all of these had received their training from Dr. Ferrier and the members of the Seattle Study Club. In passing, attention should be called to the fact that quite a number of operators had received instruction only for a short time, a matter of a few months.

The clinic was held in the Seattle Civic Auditorium, which was admirably suited to the purpose, having an abundance of light and ventilation. Lighting was by skylights with provisions for regulation to prevent the light from being too intense. The result was ideal for operating and every operator through the entire afternoon had the opportunity afforded by these surroundings to enable him to put forth his best. The equipment provided a chair and an electric engine to each operator, with ample space adjacent to each chair for observers. Opportunity was afforded observers to witness each case before operating was started, the preparation of the cavity and the completed operation, announcement being made by loud speakers as each cavity or operation was completed.

All classes of operations were done except Class 1, Class 2 and 3 predominating. I spent the entire afternoon at the clinic and, as far as possible, inspecting each finished cavity and operation. Never before at any time have I seen so much really good dentistry. Each operation was as nearly perfect as human skill and knowledge could make it, and the word "perfect" is used in the broadest sense. Perhaps the most impressive, also the best, thing about the whole clinic was the extreme conservatism displayed in the size of cavities. These operators adhere strictly to Black's principles, but apply them conservatively, never unnecessarily cutting away the slightest bit of tooth substance; the result: extremely small cavities, still with proper extension, a greatly shortened operating time, more artistic and sightly operations, less possibility of subsequent fracture and very slight chance of pulp degeneration or death.

What a fine thing if all dentists in America could have witnessed this clinic! What an education it would have provided them, what food for thought, what an inspiration to further the ends of idealism! What a beneficial effect it would have had on those inlay enthusiasts (who have boastfully stated that they have no use for gold pluggers) with their widely and deeply cut cavities! What an inspiration to those who believe in real preventive dentistry, to witness so many fine operations, which, to our positive knowledge, are the most permanent, by far, of all dental operations!

I might continue to enumerate many other fine and inspiring things which might have or did come about from this

wonderful display of professional skill and attainment.

Let each of us who believe in operative dentistry and this includes every real dentist, resolve to do his utmost to further the interest and well-being of mankind through this medium, the greatest afforded by dentistry, and in giving expression to these sentiments, his skill and artistic sense, let this be done to a great extent by the use of gold foil, the one thing in dentistry which permits of a well-nigh perfect result.

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IMPRESSION TAKING AND MATERIALS

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THEORY OF DENTAL IMPRESSIONS

DEFINITION *of an Impression.*—An impression is a suitable imprint of the desired denture-supporting and border tissues of the edentulous or partially edentulous jaw which has been obtained by properly adapting to these structures an appropriate material which retains its form on removal from the mouth.

This definition involves a knowledge of the extent, the form and the nature of the denture-supporting and border tissues and implies the use of an appropriate material to meet the tissue requirements of each impression. Herein lies the secret of good impressions.

Purpose of an Impression.—The purpose of an impression is to secure an imprint of the denture-supporting tissues which will assure, first, retention, so that the finished denture will not become loose while in use; secondly, stability, so that the denture will not move about too much during mastication, and, lastly, comfort, so that the patient may be able to use the denture.

Extent of an Impression.—The extent of an impression affects the retention of the subsequent denture, for, if the impression is overextended, the surrounding muscles of the lips, cheeks or tongue will dislodge the denture during function. Likewise, underextension of the impression will impair the retention of the finished denture because of the ingress of air between the denture and the supporting tissues, which breaks the cohesive bond of the saliva.

Form of an Impression.—The form of an impression, contrary to popular opinion, need not always and frequently should not be exactly like that of the tissues covering the residual ridge. The reasons for this variation in form are obvious when we consider that denture-supporting tissues are not always of the proper form or consistency to insure stability in the completed denture. Flabby soft tissues may require displacement, or even surgical care, while hard, nonresilient tissues may require relief of the impression in order to stabilize the finished denture.

It is important for the dentist to keep in mind that the finished impression must be stabilized on the denture-supporting tissues in order that the finished denture may function properly. The finished denture may be expected to possess the merits and faults of the impression.

*Read before the Chicago Dental Society, Sept. 20, 1932.

Further, we must not forget that denture-supporting tissues are subject to change; therefore, it is important that we consider the physiologic tolerance of the tissue while taking an impression. Failure to consider the tolerance of the tissues is responsible for premature loss of retention, which impairs the service with many newly made dentures.

IMPRESSION MATERIALS

General Remarks.—Most of the commonly used impression materials have merit when judiciously used. The secret of success in the use of any material consists in knowing when and how to use it. All operators will not have a similar degree of success in using the same material. We must learn the limitations of an impression material and select those materials which, in our hands, give the best results. Most of the failures attributed to impression materials may result from improper manipulation by the operator.

We should use that impression material which, in our hands, offers the quickest and the most simple method together with the best result. Whatever the material or method may be, I am convinced that a good examination of the denture-supporting tissues will save much valuable time in the actual taking of the impression. The average dentist should become proficient in the use of at least one material in each of the three following groups so that he may make an intelligent choice of the material to be used for each impression.

Impression Compound.—Properties and indication for use: Impression compound is a resinous base material which softens with heat varying from 120 to 150 F. and hardens at temperatures lower than about 95 F. In its plastic state, it is capable of recording the fine lines and details of the surface against which it has been pressed, and it retains an accurate imprint if it is allowed to harden prior to its removal. Compound, in its plastic state, must be pressed against the surface to be recorded. It tends, therefore, to cause displacement of tissues, especially when they are flabby. This property of compound recommends its use especially for impressions that require displacement of soft tissues in order to secure stability in the finished denture. The amount of displacement may be controlled by the plasticity of the compound and other conditions beyond the scope of this paper.

I wish to point out particularly the fact that compound is plastic, and not elastic. It flows in whatever direction it is forced, but does not flow back when pressure is released. It must be reinforced by a tray and held in contact with the desired surface until it has hardened. In the meantime, it continues to flow at a decreasingly rapid rate until it becomes hard or until the pressure is discontinued.

Owing to this property of compound, it is advisable to build up an impression in sections which can be readily controlled rather than to try to obtain the whole impression at one operation. When localized areas of soft tissue are present and are likely to impair the stability of the denture, these should be disregarded until the impression nears completion, at which time additional compound may be added to cause displacement of the soft tissue areas.

The most undesirable feature in the use of compound lies in the fact that it must be heated before it can be used. When this heated mass is applied, the tissues immediately become engorged with blood. Many operators have resorted to icewater in order to chill and to control the flow of compound, but, from my personal experience, I am inclined to regard this practice as detrimental to accurate

impressions. Icewater causes contraction of the surface capillaries so that the continued heating and chilling of the tissues soon results in swelling and reddening of the mucosa, which is thus considerably thicker by the time the impression is completed than it was when the impression was begun. This practice may be responsible for the lack of retention in some newly made dentures.

In order to avoid at least some of the faults of this method I recommend that the lowest temperature consistent with good results be employed, that every possible step of the impression be taken out of the mouth on a study model, and that ordinary tap water, if any, be used for chilling the impression in the mouth.

Plaster of Paris.—Properties and indications for its use: Plaster of Paris is calcined gypsum which when combined with water crystallizes and becomes hard. It is applied to the tissues while it is in a liquid or semiliquid state and flows into the minute inequalities of the surface, thus producing an accurate impression with the least possible amount of tissue displacement. Plaster must be held in contact with the tissues until it has crystallized, after which it may be removed without danger of changing its shape, for it will fracture rather than become distorted.

In some respects, plaster is ideal for dental impressions: It may be used at a temperature lower than that of the mouth, the consistency may be controlled to produce less tissue distortion than any other impression material, little equipment is required in its use and the impression may be quickly taken—a desirable feature with some patients.

Plaster is especially recommended for impressions of hard or nonyielding tissues and may be quite satisfactory for most impressions which do not require displacement of the tissues. It cannot be used with success where the oral tissues are quite yielding, since plaster of the proper working consistency is not resistant enough to cause displacement of the soft tissues.

Plaster of Paris has some unfavorable properties, namely, the taste, to which few patients object, and the untidiness associated with its use, which may be obviated by careful handling.

Owing to its liquid condition, plaster of Paris is most conveniently used in thin layers applied to closely fitting trays which have previously been made to conform to the tissue. This feature will be explained more fully under procedures in impression taking.

Elastic or Resilient Impression Material.—Properties and indications for its use: This type of newly developed impression material includes products such as "dentocoll" and "ke-2-ol," which have been made primarily to facilitate the taking of impressions of undercut areas, particularly in partial dentures. Most of us who have had experience in plaster and sectional-compound impressions of partially edentulous ridges will welcome this new, although incompletely developed, impression material.

I have used the terms "elastic" or "resilient," since these materials have the property of springing back to their original shape when they have been distorted. This elastic quality in an impression material, which is not possessed by either compound or plaster of Paris, is so desirable that elastic impression materials will undoubtedly be more widely used in the future.

Elastic materials are heated in boiling or hot water, and kneaded, either in a suitable mixing device, or by hand, until the desired consistency has been obtained, after which they are applied to a suitable tray and inserted.

Dentocoll requires an expensive mixing cylinder, heating vessel and trays; which will no doubt prevent wide distribution of this material. The plastic state in which this material is used requires a tray large enough to prevent overflow while the impression is being taken. Large trays, although ideal for partial impressions, interfere with peripheral adaptation of impressions for full dentures.

The use of icewater spray to chill dentocoll is rather trying to both operator and patient. New trays have been designed to overcome this disagreeable feature, but the expense will probably curtail their use.

Present development in simplifying both the method of using and the equipment for manipulating elastic materials is seen in ke-2-ol, which has been used successfully by many operators. This material, which is manipulated in the same manner as is compound, is applied to the tissues in a standard impression tray.

Elastic impression materials, which have come to stay, will undergo further improvement. Even in their present condition, they are capable of recording impressions of amazing accuracy and detail, while their elastic property permits an imprint of undercuts which was possible only through unusual skill and effort when either plaster or compound was used.

PRACTICE IN IMPRESSION TAKING

Manipulation and Use of Impression Compound.—Having discussed the properties and uses of the three types of impression materials, I desire to outline a method for taking full impressions which has been used successfully by students in the department of clinical prosthesis in the university.

When the patient first comes in, a thorough examination and diagnosis of the conditions are made. A preliminary impression of plaster or compound is taken and a study model is poured of cast plaster. Thin aluminum trays with detachable handles are adapted to the study models and trimmed, when necessary, to the desired size, after which compound is applied to the tray and an impression of the study model is taken. This tray and compound is carefully oriented to the model so that the compound is about 4 mm. thick. Excess compound is trimmed from the periphery of the impression prior to insertion.

The flame of the alcohol blowtorch is now directed against the seating surface (only) of the impression, after which the heated compound is tempered in water at 140 F. and inserted. It is seated snugly on its respective ridge (the upper is seated with upward and backward pressure, while, on the lower, the pressure is directed downward and forward). No attempt is made to muscle trim at this stage. After a few moments, the impression is carefully removed without chilling, and is submerged in a tray of water to harden.

Peripheral adaptation is now secured by separately heating suitable sections of the impression border, after which that portion is tempered in hot water, inserted and adapted by rolling the tissue of the cheeks or lip upon it until the desired peripheral contour has been obtained. This sectional adaptation is continued until the entire peripheral border of the impression has been adapted to the requirements of the soft tissues surrounding the impression. Additional compound may have to be added, or some may have to be trimmed away before this process is complete.

I desire to call attention, first, to the postpalatal border of an upper impression and, secondly, to the sublingual borders of a lower impression. The postpalatal border is the most important and

most difficult portion of the upper impression to record. In securing the correct adaptation to the pterygopalatine (posttuberosity) fossae, I have found it advisable to use the tip of the finger in adapting the compound in those areas where it is not likely to flow of its own accord. The softened compound may also be neatly adapted to the postpalatal margin by this method which insures a better adaptation along this important margin.

The sublingual borders of the lower impression are handled in precisely the same manner. I have found the tongue an unsatisfactory assistant in determining the proper extent of the lower denture. It is perhaps better to extend the lower denture as far as may be comfortably tolerated than to invite loss of retention by underextension. Overextension can always be corrected by trimming, but underextension requires a rimming operation.

Manipulation and Use of Plaster of Paris.—The foregoing procedure in impression taking applies more particularly to compound, but may be combined, in a slightly less elaborate form, with plaster-wash or correctible-compound impressions. When a plaster impression is desired, a compound tray is prepared as previously described, with the exception that the peripheral margin is not so accurately adapted to the border tissues.

The compound peripheral margin of the impression is then trimmed with a sharp knife until sufficient space is provided in which to secure a peripheral adaptation of plaster. The postpalatal margin of the upper and the lingual margins of the lower impression should not be trimmed away with a knife after they have been properly adapted.

In addition to reducing the peripheral margin of the compound tray prior to taking a plaster impression, it is advisable also to trim away any areas which may require relief in the finished impression, thus providing a thicker layer of plaster in the area requiring relief.

When the compound tray is completed, a thin mix of plaster is prepared and applied to the tray by the spatula. The tray should be covered by a thin and uniform layer of plaster, after which it is quickly carried into the mouth, oriented to position, vibrated gently to place and supported with a little pressure until it begins to crystallize. At this time, the peripheral adaptation is perfected by massaging the cheeks and lips about the buccal and labial borders of the upper or lower impression. No attempt is made to muscle trim the postpalatal or lingual margins of plaster impressions.

When the plaster has crystallized, the impression is removed. It is allowed to dry for a few moments before it is prepared for pouring. Any areas which require relief may be altered by scraping away the plaster to the desired depth. A peripheral seal may be added to the upper by flowing liquid wax (which has been heated in the bowl of the wax spatula) about the margin of the impression just inside the periphery, in order not to interfere with the movable border tissues. A single and uniformly narrow band of wax may be applied to the entire margin of the upper impression except the postpalatal, which may require two thicknesses, this depending on the depth of the yielding tissues. The lower impression may be treated in the same manner with a single layer of wax across the posttubercle margin only. This seal, when properly applied, greatly aids retention of the denture and does not in any way injure the tissue. In reality, it compensates for the expansion which occurs in many of the cast materials during vulcanization.

Before leaving the subject of plaster of Paris, I desire to mention the use of so-

called "soluble" plaster, such as "complaster" and "solvite." These materials, which have become widely used in partial impression work, possess a degree of plasticity not found in ordinary plaster of Paris. This plastic property of "soluble" plaster provides a body of material which may be easily adapted to the tissue. It is pleasant to the taste and is less difficult to remove from the mouth. I question the advisability of boiling this impression material from the cast owing to the effect which boiling may have on the material of which the cast is made. "Soluble" plaster, when similarly treated, is more readily removed from the cast than ordinary plaster.

Manipulation and Use of Dentocoll.— Preparatory to the use of dentocoll, the remaining teeth with which it may come in contact should be well cleaned and covered with a film of cocoabutter or petrolatum. The lips should likewise be protected by a suitable lubricant.

A cartridge of dentocoll is placed in the mixing cylinder together with enough water to eliminate the air from the barrel, after which the cylinder is placed in boiling water for not less than four minutes. It is then removed and thoroughly mixed with the plunger, the temperature meanwhile being reduced by submerging the barrel of the mixer in tap water. When the material has been reduced to a temperature of about 135 F., it is forced from the mixer onto a special tray which has been prepared by first applying a little of the mixture with the finger to fill the perforation and thus prevent the dentocoll from coming loose from the tray. The dentocoll is made to conform approximately to the shape of the surface to be covered, after which the surface is lubricated with a thin film of petrolatum applied by the finger. The mouth is now chilled with cold water and the dentocoll is inserted, although quite plastic, and seated in place.

Immediately, the impression tray is subjected to a continuous stream of icewater, which is maintained by using two water syringes alternately. It is advisable to have an assistant in this procedure. A suitable pan is provided and held tightly under the patient's chin to catch the outflowing icewater. About a quart of icewater is needed to thoroughly chill an average impression, which requires about four minutes' time. When chilled, the impression may be removed with the usual care and kept in a moist chamber until it is ready to be poured.

TESTING THE QUALITIES OF AN IMPRESSION

Much time can be saved and disappointment avoided by testing the retention and stability of a proposed restoration before it is completed. This can be done most effectively by preparing a permanent denture base of vulcanite or metal which may be tried and tested in the mouth. If the base is found to lack the necessary features, it may be used as an impression tray for a new impression. If the base is acceptable, it may then be used for the subsequent steps in denture construction, and finally it may be incorporated in the finished denture.

SUMMARY

1. A knowledge of denture supporting tissues is a prerequisite to impression taking.

2. The secret of success in impression taking consists in understanding the properties of an impression material and also in knowing when and how to use it.

3. Three types of impression materials are to be considered:

(*a*) Impression compound, a plastic material by which displacement of denture supporting tissues may be secured.

(*b*) Plaster of Paris, a semiliquid material which records the surface with little distortion of the tissues.

(*c*) Elastic materials, which are especially suitable for undercut areas, since they are capable of assuming their original shape after being distorted during removal of the impression.

4. A permanent denture base is recommended as the most accurate means of testing the qualities of an impression prior to finishing the denture.

PERIODONTAL DISEASES*

By WARREN WILLMAN, B.S., D.D.S., Chicago, Ill.

FOR many decades, gingival diseases have been the object of much study and the subject of endless discussion. Even today, they are usually regarded as complicated and confusing. Nearly every serious student of periodontal disturbances seems to have coined a name, usually a descriptive one, to fit his conception of what the laity and much of the profession still call pyorrhea. In the pioneer days of American dentistry, it was called "Rigg's disease," after one of the first Americans to describe it, and it is said first to have been called "pyorrhea alveolaris" by Toirac, in 1823. This term has survived more than fifty efforts to supplant it, and in spite of the fact that virtually no one welcomes it and that it always requires fresh defining and modifying whenever it is used. "A flow of pus from the tooth socket" is its literal meaning, which cannot be taken seriously today, but if it is amended to mean a flow of pus from the gingival crevice, it can be pressed into good service.

Although this disease has been observed and treated for centuries, it has only now, in the last decade, begun to be clearly understood, and while volumes have been and are being written about it, most of the literature either ignores the recent findings of research or else presents them exclusively, and thus the general practitioner has had meager pickings in his effort to find an explanation of the problems that puzzle him in this branch of his practice.

NORMAL HISTOLOGY

Two conditions have contributed largely to the confusion of former days: (1) a mistaken conception of the epithelial attachment and (2) the difficulty of discriminating between two separate diseases, which has led to their being described always as one. By 1921, Gottlieb[1] of Vienna was enabled, with the aid of the modern histologic technic, to obtain accurate specimens of human teeth, sectioned with their surrounding soft and hard tissues, in their true relationship, and from these, he discovered the nature of the epithelial attachment. Before that, it was believed that the epithelium covered the inside of the

*Read before Danville-Champaign District Dental Society, Oct. 20, 1932.

1. Gottlieb, Bernhard: Tissue Changes in Pyorrhea, J.A.D.A., 14:2178 (Dec.) 1927, Traumatic Occlusion, ibid., 14:1276 (July) 1927; Formation of Pocket; Diffuse Atrophy of Alveolar Bone, ibid., 15:462 (March) 1928.

"free gingivae" and normally terminated at the bottom of the gingival crevice in a mere line of cells, apposed to the tooth at the cemento-enamel junction (Fig. 1, left). Gottlieb was able to show that former specimens were misinterpreted, that the empty space indicating the position of the enamel (which is lost in histologic specimens by decalcification) had been interpreted as the gingival crevice, and, as a consequence, the bottom of the crevice was always stated as being at the cemento-enamel junction. By means of the newer specimens, Gottlieb showed that the epithelium is always attached to the surface of the tooth in the form of a collar or band, the upper edge of which establishes the bottom of the gingival crevice (Fig. 1, right).

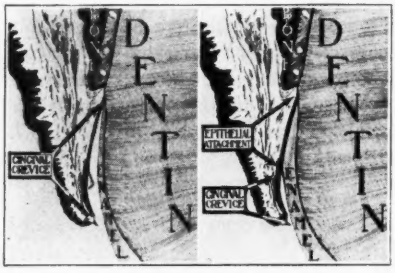

Fig. 1.—Camera lucida drawing of lingual side of an upper cuspid. Left, former conception of epithelial attachment. Cuticle is seen about midway between the crest of the free gingiva and the supposed bottom of the gingival crevice. Right, present conception, with cuticle replaced on enamel, at true bottom of gingival crevice.

It was further shown that when the tip of an erupting tooth first punctures the oral epithelium, the epithelium covering the entire enamel crown (the former enamel organ) does not, as used to be believed, immediately detach itself down to the point of the cemento-enamel junction, leaving a gingival crevice as deep as the crown is long (Fig. 2 *a*). On the contrary, this detachment proceeds more slowly, with the free gingival crest following it fairly closely. This means that, at the first moment of eruption into the mouth, we have an epithelial attachment girdling the tooth that is as wide up and down as the enamel crown is long (Fig. 2 *b*). The lower extremity of the epithelial attachment is at the cemento-enamel junction, while the upper extremity reaches almost to the tip of the cusp. (For simplicity, the term "lower" is used to mean rootwise and "upper" to mean occlusalwise, throughout this discussion.) This means an extraordinary width of epithelial attachment, a dimension which is never again duplicated during the life of the tooth. As the upper extremity of the attachment detaches itself bit by bit (Fig. 2 *c*), the lower remains stationary until most of the enamel crown has become exposed to the mouth, and the epithelial attachment attains a normal width (Fig. 2 *d*). Just how wide the normal is, no one can say. It varies with individu-

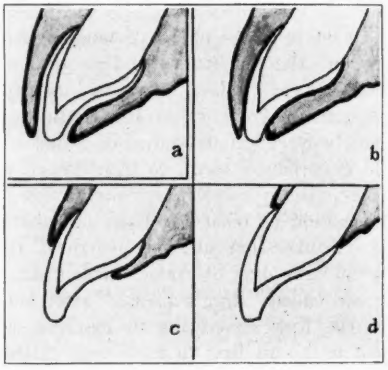

Fig. 2.—*a*, former conception of formation of gingival crevice at earliest time of eruption. *b, c, d,* progressive stages in eruption of tooth with gradual reduction in width of epithelial attachment. The constant shallow gingival crevice may be noted.

als, with different teeth, and even at different points around any one tooth, but in a very general way, it would probably average about 0.5 mm. Having once attained its narrow minimum, this band will not detach farther at its upper extremity until a corresponding or preliminary growth rootwise occurs at its lower extremity along the surface of the tooth. In this way, it maintains itself as a band of definite width and does not shrink to a mere line.

Although there is still some discussion about it, it is now quite well established that human teeth erupt continuously throughout life, and not just until they first come into occlusion with their antagonists. It has long been known that the teeth of rodents erupt continuously, but it has only recently been learned that human teeth do the same. The difference lies in the fact that human teeth erupt very slowly—only a few millimeters in a lifetime. It was formerly thought that eruption was "completed" at about 12 years of age, when all but the third molars came into occlusion with their antagonists, and that the teeth stood fixed and immovable in the jaws after that. The rate of eruption certainly is slowed to something near the infinitesimal from then on, but there is considerable and definite evidence, both clinical and microscopic, that eruption continues at varying and uneven rates for the rest of the individual's life.

Further, this eruption is twofold, or as Gottlieb very recently defined it, active and passive.[2] In the active sense, the teeth do literally erupt out of the mandible and maxilla toward the occlusal plane, carrying alveolar bone and periodontal membrane with them, but they also may be said to erupt passively into the mouth while they are standing relatively still in the jaws by means of the epithelial attachment shifting rootwise, thus exposing more of the teeth to the oral cavity. The term "clinical crown" has been devised to describe the part of the tooth that lies free in the oral cavity, above the bottom of the gingival crevice. Very early in life, therefore, the clinical crown consists of only a part of the enamel covered crown. Later on, as the tooth erupts, all of the enamel will be exposed, and still later part of the cementum will be included as a component of the clinical crown. It must be emphasized that this migration of the epithelium rootwise does not necessarily occur evenly around the entire circumference of the tooth at all times. Every clinician is familiar with the picture of the labial surface of a cuspid or the lingual root of an upper first molar exposed far in advance of the rest of that same tooth. Even more, it must be emphasized that the cemento-enamel junction does not represent a stopping place for the bottom of the crevice. The epithelium has no more tendency to remain there than at any other given point on its journey rootwise. Probably, it rarely, if ever, happens that the bottom of the gingival crevice coincides exactly with the cemento-enamel junction all around one tooth at the same time.

With this brief résumé of the normal histology and physiology, it should be remembered that, for the present discussion, the "free gingiva" is defined to mean the lip of tissue surrounding the tooth that extends above the upper border of the epithelial attachment; that the "gingival crevice" is the space (potential) between the free gingiva and the tooth, limited always by the epithelial attachment below, and, therefore, that the bottom of the gingival crevice and the upper

2. Gottlieb, Bernhard and Weinmann, J.: Paradentalpyorrhöe und Alveolaratrophie, Fortschr. d. Zahnheilk, 9:394 (May) 1933.

edge of the epithelial attachment are always one and the same. One other term will be used: the "crevice epithelium" is the epithelium that lines the inside of the gingival crevice.

HISTOPATHOLOGY

Ordinary pyorrhea, often termed marginal pyorrhea, makes up approximately 90 per cent of all clinical cases. To understand the disease and to diagnose and treat it intelligently, a knowledge of the mechanism by which it comes about is essential. Starting with the normal gingival crevice (Fig. 3 *a*), extremely shallow and lined with intact crevice epithelium, the very first step is the introduction of a slight mechanical irritation. This may be from a badly fitted crown or inlay, an overhanging amalgam filling or a food impaction resulting from a faulty contact point, but in most cases, it will result from the presence of calculus. The calculus fills the gingival crevice and causes irritation by its presence (Fig. 3 *b*). The response is a slight chronic inflammation. This is characterized by a perceptible red tinge at the border of the free gingival margin, especially noticeable in the interdental papilla, and by a slight hypertrophy (Fig. 3 *c*) of the soft tissue. The latter is of great importance because any increase in the size of the free gingival margin automatically increases the depth of the gingival crevice. The crevice, being deeper, allows the accumulation of a little more calculus (Fig. 3 *d*), which, in turn, increases the inflammation, including the hypertrophy, making the tissue side wall of the crevice higher again and allowing still more calculus to form. Worse than this, the increasing inflammation brings an increase of tissue fluids to the part and so creates an increase in the fluid pressure of the region about the gingival margin. The crest of the alveolar bone, being extremely labile to the slightest change in pressure soon becomes honeycombed at its upper border by osteoclastic resorption (Fig. 3 *e*). (This gives rise to the granular or "necrotic" feeling of the bone margin under a surgical instrument, as often observed by exponents of heroic surgical treatment for pyorrhea. Actually, of course, there is no necrosis or caries or even infection of this alveolar margin, because the resorption is taking place under the covering of the mucous membrane.)

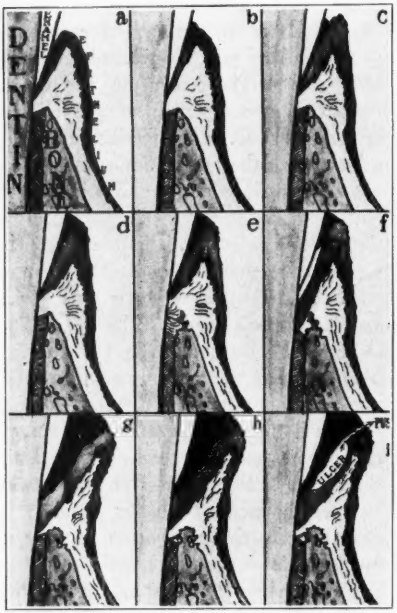

Fig. 3.—Schematic representation of successive steps in advance of marginal pyorrhea.

Before the next step is discussed, a curious phenomenon must be noted. The epithelial attachment has a tendency, as yet unexplained, to grow rootwise along any tooth, additional areas of root surface being thereby exposed. In this re-

spect, it treats a tooth much as an implanted foreign body. This singular biologic tragedy is normally averted by the presence of the live periodontal membrane fibers stretched from the tooth to the crest of the alveolar process, directly across the path of the down-growing epithelium. While these fibers are present and functioning, the epithelium cannot possibly invade the space they occupy.

As the alveolar crest is resorbed, owing to the increase of fluid pressure in the tissues about it, the uppermost fibers of the periodontal membrane are released (Fig. 3 *e*) and cease to function. When this occurs, the epithelium is enabled to grow or extend its attachment rootwise, and since it seems bound by some obscure law to maintain a fairly uniform average width, it detaches from the tooth surface a corresponding distance above. In other words, the epithelial attachment migrates bodily down the side of the tooth as soon as a few periodontal membrane fibers are gone (Fig. 3 *f*). This amounts to a premature eruption of the tooth a little way into the oral cavity. The bottom of the gingival crevice is now at a slightly lower position on the tooth surface, and, consequently, the gingival crevice is even deeper than before, admitting of a further deposition of calculus (Fig. 3 *g*).

In this way, a vicious circle is established. Calculus causes inflammation with a hypertrophy of the gingival margin; which allows more calculus to be deposited. The resulting inflammation causes enough increase in tissue fluid pressure to initiate a little resorption of the crest of the alveolar process; which severs a few of the uppermost fibers of the periodontal membrane, throwing them out of function. This permits the epithelial attachment to descend along the side of the tooth, and thus the crevice deepens and more calculus is deposited, the whole process being repeated in a more exaggerated tempo. As long as this process continues, the crevice becomes deeper by extension at both ends: by hypertrophy of the free gingival margin at the upper border and by a downward growth of the epithelial attachment at the lower. If left to itself, this process goes on until the crevice becomes so deep that it is convenient for descriptive purposes to change its name and call it a pocket. Just when a deep gingival crevice shall be known as a pocket is as arbitrary and indefinite as the point at which a snowstorm becomes a blizzard. It remains a valuable and necessary clinical distinction and is not necessarily so vague as might be thought. The presence of the pocket is the *sine qua non* of all pyorrhea; for without it pyorrhea cannot exist.

The pathologic changes described up to this point are those of chronic gingivitis, the invariable precursor of marginal pyorrhea. Now, with the chronic inflammation greatly prolonged and exaggerated and the crevice deepened to the proportions of a pocket, a new factor enters the picture. The crevice epithelium, insulted for so long by the mechanical irritation of the adjacent calculus and injured by the action of the omnipresent bacteria of the mouth (Fig. 3 *h*), finally succumbs to the attack of the latter and an ulcer is formed (Fig. 3 *i*). An ulcer is defined as a superficial circumscribed area of suppuration, and, with the first drop of pus that is elaborated by this ulcer, pyorrhea in the true sense of the word begins. The flow of pus comes, therefore, not from the periodontal membrane or from the alveolar bone, as was once believed, but from the oral cavity itself, that is, from that part of the mouth epithelium that lines the inside of the gingival crevice, namely, the

crevice epithelium. If the ulcer were on the cheek, opposite a jagged broken tooth, it would be easily accounted for, and its being on the side wall of the gingival crevice, opposite rough calculus, is just as easy to understand. If it is not interfered with, the pocket goes on getting deeper, the inflammation increases, the ulcer on the crevice epithelium enlarges and secretes more pus, the bone destruction becomes greater, and, finally, the tooth becomes loose from loss of bony support and eventually falls out. Broadly then, simple marginal pyorrhea divides itself into three major phases: inflammation, the production of the pocket and the loosening of the tooth.

But what of the constitutional factors, and that other 10 per cent of clinical cases that are so baffling? These have certainly been the cause of most of the confusion in the field of periodontia. And no wonder. Suppose tuberculosis rarely occurred except in conjunction with pneumonia: would it not certainly be tangled up clinically for years as an unusual type of pneumonia? A similar situation has existed in the case of gingival disease. Many types and kinds of pyorrhea have been described and ascribed to divers sources because two diseases, or mixtures of them both, were being observed. The first, marginal pyorrhea, of local origin, has been described. The second has been termed, for reasons that will be apparent later, diffuse atrophy of the alveolar bone. In the progress of this fairly rare disease, the cardinal features of marginal pyorrhea, i. e., inflammation, pocket formation, and loosening of the teeth, are presented in exactly the reverse order. The very first manifestation of diffuse atrophy in a clearly marked, unmixed case is a loosening and wandering of the teeth. As I have often stated to students, it is as though an "invisible orthodontia appliance" were operating on them. One tooth, group of teeth, or all the teeth may be attacked at once; there is no rule. The teeth may move in any direction except back into their sockets. If they move into such a position that they receive additional and unusual punishment from their antagonists, they are said to be in "traumatic occlusion," but it cannot be too strongly emphasized that occlusal trauma is not the primary cause of any periodontal disease. It is never more than a link in a chain of pathologic events. In the case just mentioned, it is secondary to the tooth movement which permitted the trauma to start; the movement itself is the primary disturbance.

It is a rare privilege to discover a clear case of diffuse atrophy in the first stage when loosening and wandering of the teeth have commenced without any abnormality of the gingival crevice. Usually, the second feature, pocket formation, has occurred before the patient applies for treatment. This pocket formation is seemingly spontaneous and is unlike that of marginal pyorrhea. The pocket appears quickly and is narrow and sharp in the direction of the long axis of the tooth. The term "vertical pyorrhea" was derived from this feature. It appears characteristically on the side from which the tooth is moving, and when it is first produced in an uncontaminated case of diffuse atrophy, it is free of calculus or detritus; consequently, there is no inflammation present, and, of course, no pus. This finding has given rise to bizarre terms such as "dry pyorrhea" and "pyorrhea without pus."

Even in a mouth with no deposits of calculus whatever, this second phase cannot continue for long. The pockets have appeared, even though not through an inflammatory process as before, and immediately bring a pyorrhea in their wake.

Food remnants, if nothing else, will accumulate in the pockets and cause an irritation with a resultant inflammation. Eventually, this will proceed to the point of a breaking down of the resistance of the crevice epithelium and an infection of it by whatever organisms are at hand, with the production of an ulcer. By this time, the clinical picture of diffuse atrophy so nearly resembles an advanced case of ordinary pyorrhea that the two diseases have been confused for generations. It is still customary to consider them as two types of pyorrhea. Some authors even term diffuse atrophy "true pyorrhea," perhaps because it is a bit more mysterious and spectacular from the viewpoint of the pathologist than a mere exaggerated irritation arising from prolonged neglect of oral hygiene.

At all events, these diseases have already been enough named and renamed to discourage any new attempt. But, for the purpose of getting a clear understanding, it seems wise to consider these two types of gingival disturbances, for the time being, as two utterly distinct diseases, just as tuberculosis and pneumonia are entirely separate diseases of the lungs.

To continue: One can easily imagine that if all advanced cases of tuberculosis resulted in the patient's being so weakened that he lay motionless on his back and so developed hypostatic pneumonia, the medical profession could easily confuse the two diseases and regard tuberculosis as a "type" of pneumonia, especially if the latter disease were clinically common, and the former quite rare. The dental profession in just such a manner has long been confused because diffuse alveolar atrophy, a comparatively rare disease, commonly concludes with an inflammation and subsequent suppuration of the pockets it produces, so that we have a "hypostatic pyorrhea," figuratively speaking, superimposed on the diffuse atrophy. That is to say, diffuse atrophy would be quite capable of destroying the entire periodontium by its own processes and their mechanical sequelae, without the help of this incidental pyorrhea, but, in actual cases, it practically never has the opportunity to do this.

Of the mechanism of diffuse atrophy, far less is known than in the case of marginal pyorrhea. It still lies rather largely in the realm of speculation; but, with what is known, a good deal may be deduced by a process of elimination.

It is known that cementum is laid down on the root surface of the teeth throughout life. When laid down in large amounts, it is often termed hyperplastic cementum and the process is referred to as hypercementosis, and relegated to pathology. This is surely erroneous. Cementum is physiologically added to former cementum throughout life, and exceptionally large deposits only indicate exceptional needs and a vigorous response on the part of the cementoblasts. In other words, cementum hyperplasia is desirable whenever it occurs. Just as the eruption of the teeth in an ideal denture compensates for the shortening of the teeth by abrasion, so the addition of new layers of cementum compensates for the reduction of practicable root length by creating a greater amount of root surface.

For this present consideration, cementum production serves another most important service. With but few exceptions, all the cells and fibers of the body die and are replaced many times during life. The periodontal membrane fibers are no exception to this general rule. They are believed, in fact, to be replaced rather frequently. When an old fiber or bundle of fibers die off, the new ones that take their place must be anchored securely

in both alveolar bone and root surface. On the parietal side, this is readily accomplished by resorption and repair of the alveolar bone, a process which is occurring constantly and thus is quite regularly observed in specimens of these tissues. Since, on the root surface, resorption and repair do not ordinarily occur, the inner ends of the new periodontal membrane fibers must be attached to the tooth, if at all, by being cemented there with the next deposition of cementum.

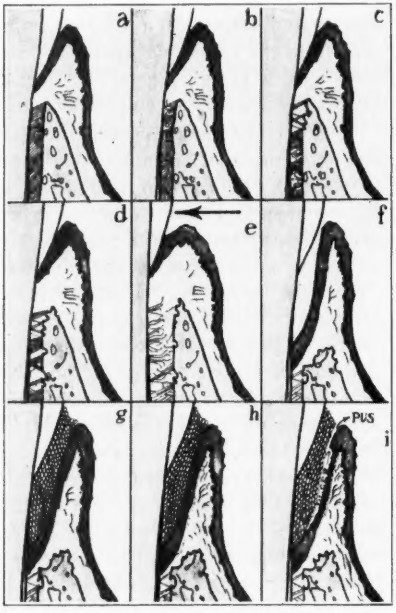

Fig. 4.—Schematic representation of probable steps in development of diffuse atrophy of alveolar bone, according to one hypothesis.

So much is fairly well established. To explain the phenomena of diffuse atrophy, it has been suggested by Gottlieb that probably the first step from the normal (Fig. 4 *a*) is the cessation, for reasons entirely unknown, of cementoblastic activity. No new cementum is laid down. As soon, then, as new periodontal membrane fibers are elaborated to take the place of senescent ones, they will be wanting attachment to the root surface. They may be embedded in the alveolar bone, but, failing a new deposition of cementum, they will be fastened at but one end (Fig. 4 *b*). If this state of inactivity of the cementoblasts persists, the alveolar bone will soon atrophy for lack of functional pull on these fibers (Fig. 4 *c*), since it is well known that the alveolar bone is very labile or sensitive to even slight stimulation. As soon as many of the periodontal membrane fibers about any tooth or teeth are of the new and unattached generation of fibers, the atrophy of the alveolar bone will become quite general; hence, the term "diffuse alveolar atrophy" (Fig. 4 *d*).

From a mechanical standpoint, the disease is predestined even more than marginal pyorrhea to run into a vicious circle. The suspensory ligaments of the tooth, i. e., the periodontal membrane fibers, are supposed to keep the tooth under all stresses from striking the side walls of the bony alveolus. When this apparatus is weakened by disease, sooner or later the normal stress placed laterally against the tooth will push it violently against one side of the socket, crushing the bone slightly. This injury of the bone would result in resorption, and in repair if it were not soon repeated; but, in this case, as the overstretched ligaments are more likely than before to permit the same damage, the bone on the side of pressure is broken down farther before repair can take place. When it is gone, the already thinned and enfeebled periodontal membrane fibers on the side of pull are soon torn off entirely (Fig. 4 *e*). Then following its familar tendency, the epithelial attachment rapidly grows

down the root surface, the typical deep, vertical pocket resulting (Fig. 4 *f*) on the side of pull, i. e., the side from which the tooth is moving. Thus, a bad cycle is established, with the damaged periodontium losing ground more swiftly as the tooth becomes looser. If, in addition, the movement of the tooth happens, as it often does, to be in such a direction that it comes into an abnormally close relationship with its antagonist, occlusal trauma steps in to carry the process to a speedy end; and since the presence of food and other irritants (Fig. 4 *g*) in the newly formed pocket invites inflammation (Fig. 4 *h*) and infection (Fig. 4 *i*), it is obvious that this disease rapidly becomes grave.

In diffuse atrophy, then, it is thought that the teeth first become loose because of the failure of new cementum to attach new periodontal membrane fibers to the teeth; the pockets develop as soon as the remaining old fibers are torn away to allow the epithelial attachment to pass down toward the root ends; and inflammation and suppuration develop at last and only because the opportunity is well presented. They have nothing directly to do with the process.

DIAGNOSIS

While the pathologic or theoretic background is essential for an understanding of these diseases, the practitioner and the patient are always most concerned with the diagnoses and prognoses. Clinically, about 90 per cent of cases of periodontal disease are unmixed marginal pyorrhea due to local irritation. This is nearly always caused directly by calculus and, for that reason, is usually seen only toward middle life, since deposits of calculus are usually just perceptible after 25 years of age and increase thereafter but very slowly. Thus, up to 35 or 40 years, there is ordinarily only an increasingly severe gingivitis. (Occasionally, of course, there are peculiar jagged deposits on the teeth of adolescents that will cause a violent gingivitis or even genuine pyorrhea. They are not at all dense or hard to remove, but are extremely irritating to the young gingival tissues. Every clinician sees these cases occasionally.) Diffuse atrophy complicates about 10 per cent, it is estimated, of clinical cases. It is rarely seen alone, for the reason that it rarely seems to occur before 25 or 30 years of age, when nearly every one has some slight marginal gingivitis from deposits of calculus. Only rarely one is privileged to see diffuse atrophy at work in a perfectly hygienic mouth.

To differentiate the two diseases, let us first construct two clinical pictures representing the extreme types. For the first, we have a person between 40 and 50. His teeth are in normal alinement and occlusion and either have cavities in them or fillings representing former cavities. Calculus is deposited heavily on the roots of all the teeth. The gingival tissues are a dead purplish red, chronically engorged with venous blood and prone to bleed on slight pressure. Pus is present in small quantities at the free gingival margin and can be expressed from the pockets by a finger run over the surface of the tissue from gingival to occlusal aspect. A suitable explorer reveals pockets around many or most of the teeth, corresponding in depth to the amount and age of the deposits of calculus. The pockets are irregularly horizontal. The teeth are slightly or quite loose, according to the amount of remaining root surface below the epithelial attachment and the amount of inflammation present to induce bone resorption. Except for this loosening, the teeth are not changing position. The disease has been growing in severity very slowly for years and has been entirely

painless to the patient. In fact, he may be totally unaware that he has pyorrhea at all.

For the second picture, that of diffuse atrophy, we have a young person in the early thirties who has an immaculate mouth. He has flawless teeth without fillings and gums of light shell pink, sometimes almost pale. There is not a trace of calculus and the gingival crevices are nearly all extremely shallow and free of inflammation. But the upper incisors are drifting. They are beginning to protrude and extrude and rotate, diastemas, or spaces, forming between them. On the lingual aspect of these teeth, as nowhere else in the mouth, are deep v-shaped vertical pockets. A suitable explorer shows that they extend well down toward the apices of these teeth, but that the root surfaces so exposed are smooth and free of calculus. Sordes and food detritus fill the pockets to the despair of the patient, who probably knows he cannot clean them, and the gingival margin is very swollen and shiny, and of a vivid deep, not a purplish, red. A test of the occlusion may show that the lower incisors are biting against the lingual surfaces of the extruding upper incisors, helping to lift them forward, or again, they may not be in occlusion at all. The disease is of recent origin, and the patient is aware that the teeth are moving and diastemas are forming. In fact, that is probably why he has come to consult the dentist.

The first of these pictures, that of marginal pyorrhea, is met very frequently in practice, along with all the stages that lead up to it, but the second is fairly rare, partly because it is actually uncommon, but also because when diffuse atrophy does occur, it is very likely to be in a mouth already slightly or greatly affected by ordinary pyorrhea, simply because there are so many such mouths. In the picture of a typical case of diffuse atrophy, the teeth were mentioned as being without caries or fillings. This is so often a concomitant feature that it is a very important diagnostic sign, though by no means a pathognomonic one, as typical cases of diffuse atrophy occasionally appear in mouths with caries. Also, the disease was said to affect the upper incisors. It is probably not more liable to attack the incisors than to attack other teeth, but it is more easily seen and recognized in the anterior part of the mouth, because of the relatively greater freedom of the teeth there to drift in other directions than straight out of their sockets. For this reason, the clinician is likely to feel that he sees more cases affecting the anterior than affecting the posterior teeth. Sometimes, all the denture is included.

The intermediate clinical pictures, from the cases of marginal pyorrhea complicated by diffuse atrophy to those of diffuse atrophy in which a gingivitis has previously been present, are harder to diagnose. But if the two extremes are kept in mind, it is surprising how accurately they can be observed. As soon as teeth are found to be looser than one would expect from the amount of attachment lost and the amount of inflammation adjacent, and as soon as pockets are deeper than the amount and position of the calculus would seem to warrant, one is justified in suspecting that diffuse atrophy is present along with marginal pyorrhea. When the teeth begin actually to drift—to protrude, extrude and/or rotate—and when the pockets are long and narrow, or even excessively deep with but little calculus present, one may be sure that even though there is calculus present with some attendant gingivitis, the case is pri-

marily one of diffuse atrophy. The absence of caries, while not essential to a diagnosis, often helps greatly toward that end in a doubtful case.

A correct diagnosis of these two great "types of pyorrhea," as they are usually called, is essential, not so much as a prerequisite of successful treatment as to enable the practitioner to make an accurate prognosis. If the case in hand is one of marginal pyorrhea alone, the prognosis is usually hopeful. With proper treatment and adequate after-care, the teeth may be retained indefinitely; but the more a diffuse alveolar atrophy complicates the clinical syndrome, the more discouraging the outlook is; and if the case is one of diffuse atrophy alone, failure to save the teeth is nearly inevitable. So lest we stumble and lose patients by making the wrong kind of predictions, we must be able to discriminate between these two diseases. One is safe in promising good results in a case of marginal pyorrhea if the disease has not progressed too far, provided after-care is never neglected; but if diffuse atrophy is concerned in a case, one should be more pessimistic in dealing with the patient. If this is carried to the point where the patient becomes so discouraged that he does not return, it is probably as well; for diffuse atrophy cases are very unsatisfactory to treat, and a thorough understanding of the probable hopelessness of the case is an absolute prerequisite to undertaking treatment at all.

TREATMENT

The treatment of marginal pyorrhea is simple to state, though difficult to master. As the presence of the pocket is the prime essential for the existence of pyorrhea, the logical treatment is obviously to get rid of the pocket. This may be accomplished in one of three ways: by removing the irritant that is producing the pocket, by removing the outer wall of the pocket itself (the free gingival tissue) or by removing the inner wall of the pocket (the tooth).

The most common and conservative treatment is the first. The removal of local irritants includes (1) the removal or remedying of all incorrect dental restorations (overhanging filling margins, crown bands, badly adapted clasps, etc.); (2) the smoothing and polishing of "sandy enamel" near the cemento-enamel junction, and, most of all (3) the removal of all calculus from all tooth surfaces. The latter is quickly and easily said, but to learn how to do it is more difficult than any other phase of operative dentistry. Once learned, accurate scaling is moderately easy, but it is undoubtedly true that more cases of pyorrhea are lost because the practitioner fails to remove all the calculus from the teeth than for any other reason. Unfortunately it is much easier to prescribe a mouth wash or paint a medicament on the gingival tissues than it is to scale the teeth so thoroughly that no calculus whatever remains on them. That is why so many cases of pyorrhea are "treated" with no visible improvement.

Once the calculus and other irritants are removed, the inflammation vanishes from the gingival tissues and they shrink down to normal, shallowing the gingival crevice to a normal depth. If this shrinkage fails to reduce the pocket to a reasonable minimum, the second form of treatment, i. e., surgical removal of the free gingivae, is employed; but again it should be emphasized that the better the scaling and polishing of the teeth, the fewer the times that a knife will need to be used. (Far too often one hears the statement made that if the tissue is cut away first, the calculus is more easy of access. Such a statement surely does not

commend the digital dexterity of the operator.) After the inflammation has abated and the tissue is shrunk to a normal contour, a regeneration of the bony crest of the alveolar process may occur, if it has been resorbed owing to the pressure of tissue fluids. If the disease has progressed far enough, in the case of any individual tooth, to allow the epithelial attachment to grow rootwise, the clinical crown will be slightly longer and the functioning root portion slightly shorter than before. Most attempts to secure a reattachment of epithelium to cementum that has once been exposed to the mouth have met with little success up to the present time, so

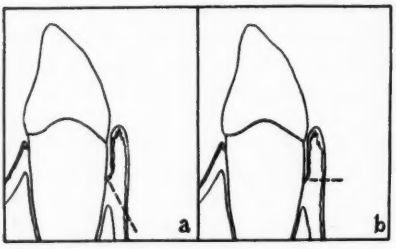

Fig. 5.—*a*, correct, and *b*, incorrect, direction of incision when free gingivae require excision.

that treatments intended to produce such results cannot be sincerely recommended. If the clinical crown (i. e., that part of the tooth above the epithelial attachment, whether enamel alone or with some cementum) has such great length after the treatment is finished that too much leverage on the attachment of the remaining functioning root surface seems probable, it should be shortened by grinding. This is frequently the case with the lower anterior teeth. In such a case, the disease has hastened the physiologic eruption of the crowns unnaturally and faster than they could possibly wear down by natural abrasion. Therefore, surgical abrasion by carborundum stones and disks has to be substituted to restore mechanical balance.

If a pocket is so shaped or situated that it will not disappear when the gingival tissue shrinks, it must be eliminated with the knife. When this is done, the cut must be in such a direction that the contour of the normal gingivae is simulated: i. e., the cut must slant sharply rootwise away from the tooth (Fig. 5 *a*). If it is made at a right angle to the tooth surface (Fig. 5 *b*), granulation tissue will quickly pile up afterward and replace most of the tissue that was removed. The tissue removed is the free gingival tissue. The cut should not extend below the epithelial attachment. Many operators who advocate heroic surgical treatment do cut below the bottom of the gingival crevice—some even go so far as to scrape or saw the "roughened" alveolar crest—but such treatment must either be the result of a desire to be spectacular or of ignorance of the histopathology of the disease. True, these cases are always a success, because a clot is formed which organizes and builds new tissue over the mutilated bone; but the resulting epithelial attachment is formed at a new level, farther rootwise than before, and the finished case, while free of pockets and infection, represents a needless exposure of valuable root surface. Since caries or necrosis or infection of the alveolar bone does not exist in pyorrhea, the only object to be gained by use of the knife is the removal of the outer or tissue wall of the pocket; therefore, cutting below the attachment is never necessary.

One exception is recognizable: when a pocket, such as an interproximal pocket between two bicuspids, is so situated that a hollow or crater is formed surrounded by healthy tissue, this tissue may require removal for mechanical reasons. The

buccal and lingual tissue, even some of the bone, might need to be cut away in order that the interproximal space could readily be kept clean. Such surgical procedure is performed for architectural reasons only, to secure proper cleansing and hygiene of the former pocket.

Whenever tissue is removed surgically, care will be necessary to prevent it from growing back to its former position. A pack or dressing in the form of cement is often valuable, if enough tissue has been removed that it can be locked in place. Otherwise, it is advisable to etch the cut surface with a 5 per cent silver nitrate or zinc chloride solution every other day for a few times. As the gingival tissues always show this tendency to rapid growth, the operator will do well to cut away slightly too much rather than too little tissue.

The third way of eliminating the pocket is to extract the tooth forming its inner wall. Although pyorrhea is entirely a disease of the soft tissues surrounding the tooth, it is conceded by everybody that extracting the tooth will stop it at once. The laity, and by natural corollary the profession, are in fact interested almost solely in saving the teeth when pyorrhea is discussed, although the teeth themselves are in no way directly involved in the disease. But when the epithelial attachment has migrated so far rootwise that the remaining functional root-end is obviously incapable of ever again supporting the tooth, or when the gingival crevice has extended into areas (such as high up on the lingual side of an upper incisor or into the bifurcation of the roots of a molar) where a pocket can never be satisfactorily eliminated for morphologic reasons, extraction is indicated.

The final phase of the treatment of marginal pyorrhea, after all irritants are removed and all pockets obliterated, is the after-care. Accurate and thorough routine brushing by the patient, using a method such as that of Charters,[3] will accomplish much by removing the early gelatinous matrix of the calculus and by massaging the gingival tissues. The better this care by the patient, the more effective and lasting the results of the dentist's treatment will be. Even so, it is a requirement that a routine prophylaxis be observed by the dentist at intervals of a few months. Every one who has engaged in this type of work knows that the case is arrested just as long and as completely as the after-care is thorough.

For diffuse alveolar atrophy, no specific treatment is as yet known. As it is not known what activates cementoblasts, there is no assignable reason for their ceasing to function. Their apparent close kinship to osteoblasts has suggested the internal administration of therapeutic doses of arsenic; but with negligible results. Arsenic is supposed to stimulate osteoblasts, but seems to have little effect on cementoblasts. Recent literature has been replete with suggestions that diet, especially as related to mineral metabolism, may be of value in the treatment of this disease, but so far no definite relationship has been shown between any phase of diet and diffuse atrophy of the alveolar bone. Up to the present, the only treatment that has proved of any service has been the purely symptomatic: the scaling of the teeth if any calculus is present; the elimination of pockets by surgical procedure when feasible, to reduce the local inflammation that augments the damage to the periodontium;

3. Charters, W. J.: Immunizing Mouth Tissue to Infection by Correct Stimulation with Toothbrush, J.A.D.A., 15:87 (Jan.) 1592 (Aug.) 1928.

the grinding of teeth that have moved into such positions that they receive undue stress, and the removal of teeth that are hopelessly involved.

It will be seen that the treatment is almost identical to that for marginal pyorrhea, and it must be reiterated that it is only intended to allay the secondary factors of inflammation and trauma. It does nothing whatever to remedy the failure of the cementoblasts to function and nothing to induce them to renew their activity. The only hope—and it is a very slender one—is that if the teeth can be maintained long enough, a spontaneous arrest may occur. This sometimes happens. The teeth drift for a time, and then suddenly stop and become firm again. They may function indefinitely in their new position, or the diffuse atrophy may start again later.

SUMMARY

Except for specific infections of the gingivae, such as Vincent's infection, and to ignore, of course, all oral manifestations of known diseases, as tuberculosis or syphilis, the actual diseases of periodontal structures may be grouped under two general phases: the slow process of marginal pyorrhea induced by the presence of calculus or by other local irritation, and the more rapid and irregular diffuse alveolar atrophy caused by unknown but probably metabolic disturbances.

Marginal pyorrhea is characterized by inflammation, pocket formation and, finally, loosening and loss of the teeth. The presence of the pocket is the most important feature, as this means exposure of an extensive crevice epithelium to continual irritation until it ulcerates and pus is produced. The elimination of the pocket is the chief aim of all treatment, whether it is accomplished simply by removing all mechanical irritants (calculus, etc.) and thereby letting the gingival margin shrink back, by excision of the free gingiva itself or by the extraction of the tooth. The prognosis is good if the disease has not progressed too far and if the treatment is thorough and the after-care complete.

Diffuse alveolar atrophy is clinically a rarer disease and of most obscure etiology. It is characterized by a primary loosening and wandering of the teeth followed by secondary pocket formation and suppuration. It is possibly due to a failure of the cementoblasts to deposit layers of cementum to anchor new periodontal membrane fibers as they are developed to replace old ones. No specific treatment is known.

THE MOUTH IN PREGNANCY*

By CHARLES B. REED, M.D., Chicago, Ill.

DISORDERS of the oral cavity during gestation are not ordinarily important in themselves, but they achieve importance as the significant signs of systemic unbalance and as vital factors in the comfort and safety of mother and child.

The tradition is still widespread that every child will cost a tooth, but only in the last year or so has it been revealed why this sacrifice is required. The subject is not only extraordinarily interesting, but it is also developing a vast and fertile field for exploration which promises to be valuable alike to the pregnant and the nonpregnant.

The main question has not been satisfactorily answered hitherto. Why, we ask, should a normal woman with good teeth and perfect fillings suffer impairment of one or both of these conditions after two or three months of gestation while diet and habits of life remain practically unchanged?

In attempting to solve this enigma, it must be noted preliminarily that a few well-known beliefs can be mentioned which have gained in repute rather than lost by scientific progress. For instance, we can still accept the opinion that proper food, in adequate amount, during the prenatal period is essential to the normal development of the permanent as well as the deciduous teeth; essential, because calcification begins as early as the seventeenth week of intrauterine life, while the crowns are, for the most part, formed before birth.

Teeth are closely related in chemical composition to the bones, and since the fetus is a true parasite, the minerals for bone and tooth formation, especially calcium, must be obtained either from food through the mother or at her expense. The calcium reaches the fetus easily by way of the placental circulation. Bar has estimated that, during the latter months of pregnancy, the fetus requires 0.638 gm. of calcium a day, which is more than the entire intake by food for most women since the usual American diet is deficient in the calcium derived from dairy products. Sherman has found that the average amount of calcium furnished by the food is 0.45 gm. per hundred cubic centimeters of protein, while a whole gram is needed to maintain the calcium equilibrium. Our people are interested in oral health but not as yet in diet. If, therefore, the food does not supply the required amount of mineral matter, the fetus will demand and take the salts of calcium and phosphorus and other materials from the blood, the bones and the teeth of the mother. This process will continue, as we shall see, until not only is the most careful dental work wholly lost through enlargement of filled cavities, but also the maternal health may be seriously disturbed, the structure of the child threatened and the pregnancy endangered. In

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this particular emergency, the mother must eat for two.

The bone trabeculae and the teeth represent storehouses of easily available calcium, and when this supply is exhausted, the shafts of the long bones are compelled to contribute.

The utilization of the calcium, taken up from within or artificially supplied, depends entirely on the vitamins C and D, which must be present in the food consumed or furnished additionally to correct the defect.

In children frequently and in adults more rarely, except under special conditions, the unsuccessful utilization of calcium and phosphorus produces rickets on account of the lack of vitamin D. While the lack of vitamin C in connection with the same deprivation of salts results in scurvy.

Pregnancy, for instance, may, by its unusual demands or imperfect utilization of calcium and phosphorus, exhibit rickets and scurvy as well as more serious diseases which reveal their earliest symptoms in the mouth as dental caries or gingivitis or both.

Let us consider briefly the calcium and phosphorus necessities for the mineralization of the fetus, which Hoffstroem says has a storage capacity at term of 30.5 gm.

Normally the blood serum carries approximately 9 mg. of calcium to each hundred cubic centimeters of serum, and the body as a whole, 3.7 mg. of phosphorus. The maternal bones contain about 85 per cent of calcium. The fetus demands 0.006 gm. of calcium a day during the first three or four months and 0.6 gm. a day at term. Bar, as well as Givens and Macy, estimates that the amount is even greater. A certain proportion of this amount could be furnished by special foods, since cow's milk gives up 30 per cent and human milk 70 per cent of its calcium.

There is much evidence (LaMer and Chown) that normal calcification depends very largely on the calcium solution in the blood being saturated. Mazzacco, Krebs and Briggs and others have observed a definite diminution of calcium in the blood of the pregnant as compared with the nonpregnant. Therefore, in spite of the increased demand for calcium in pregnancy, the deficiency is often as much as 0.42 mg. per hundred cubic centimeters.

In consequence of defects of mineralization through absence of vitamin D, dental caries ensues. The opinion is general among dentists that tooth deterioration begins on the hard enamel sheathing and works inward, hence, the unqualified statement just made will meet with much doubt and incredulity from men who have been reared on different faiths and principles, and the reasons for it must be set forth. What is the cause of dental decay?

Bacterial activity is broadly accepted and heralded as one of the sources of caries according to the chemicoparasitic theory; but definite objections appear. The fact that bacteria are always present in carious dentin or enamel proves nothing, since every cubic centimeter of saliva contains normally from 5,000,000 to 50,000,000 bacteria. Admitted, as it must be, that certain germs may traverse healthy tissue and infect distant organs, as, for example, when *B. coli* moves from the rectum to bladder or kidney, the bacteria of the mouth are not known to have this capacity. With this preamble, the report of Jones, Larsen and Pritchard of Hawaii is most significant. They review 140 cases in which many teeth were found to be decayed before eruption occurred. In these cases, there was no cell infiltration, no soft tissue reaction, no evidence of bacterial invasion in the bone crypts nor any osteoclasts. If such evidence proves anything, it is that the presence of bac-

teria must be regarded as incidental and secondary rather than causative in caries. Such evidence would show that dental caries is a chemical process in which systemic conditions are definitely potent.

Particles of food, stagnant deposits of carbohydrates, lodged between the teeth and encapsuled by mucin, may, it is claimed, form culture mediums in which micro-organisms develop and secrete an acid which injures the enamel. Under normal conditions, this theory cannot be reasonably defended because the ptyalyzing action of the saliva catabolizes the food fragments in a very short time, and the resulting sugar or débris is carried away before the bacteria or their acid products can affect the teeth. Furthermore, this process would be impossible where decay occurred in unerupted teeth. Hence, this hypothesis is apparently incompetent *per se*.

Salivary acidity and the presence of alkaline buffer substances may so vary as to be favorable or unfavorable to the production of caries, as Brooker has stated. Now calcium and phosphorus are the most important alkaline buffers which are carried in the blood or excreted by the saliva, and a diminution of these substances or a failure in utilization through the absence of vitamin D might perhaps diminish the immunity and allow the acid plaque to form and destroy the enamel. The hardness of enamel enables it to resist attack until p_H 5 is reached, but it seems hardly probable that such a degree of acidity could be produced and retained unless the alkaline elements in the blood, calcium and phosphorus, were so markedly deficient that their immunizing power was destroyed.

The parathyroid glands, according to Falta, receive unusual demands in pregnancy and, in their response, a latent deficiency of calcium is unmasked. The physiologic function of the parathyroids is to regulate calcium metabolism and maintain it at a definite level in the blood. This office is accomplished by withdrawing calcium from the spongiosa of the bones for systemic utilization. The calcium thus borrowed is not returned, but is excreted through the bowels and kidneys. In a way, this endocrine theory is important since the effort of the glands to restore a depleted element decalcifies the teeth and so reduces the immunity as to permit the assaults of local, possibly acid, agents. Though parathyroidectomy results in hypocalcemia, statements regarding the effect of the operation on the structure of the teeth have not thus far come to our attention.

The most generally accepted theory of the cause of caries in pregnancy, and indeed elsewhere, assumes that the oral secretions become extremely acid and, as a result, the surfaces immersed in saliva or merely moistened therewith will break down. Milton says that the caries of dentin is much more rapid than that of enamel, and that the process removes both calcium salts and inorganic matter. He holds also that caries of enamel can be produced by very dilute acids. The statement as presented is not convincing.

The teeth are composed of inorganic material protected by a flinty enamel which makes them chemically inert. They occupy a moist sterile chamber subject to wide variations of temperature, atmospheric gases and contact with food products. If, as postulated, dental caries consists of localized areas wherein the mineral constituents are dissolved by oral acidity or enzymes, the entire denture must be equally exposed to the action of eroding acids; and yet, most frequently, only one tooth breaks down on its buccal or lateral face, while its contacting neighbor is unblemished. Such a process does not seem to be chemically plausible unless the theory of plaques is sustained.

The normal saliva, at p_H 7.1 to 6.6, is neutral or mildly acid, and while this acidity, of course, may be increased at times, the change is neither sufficiently frequent nor sufficiently intense to account for the occurrence of caries either in the pregnant or in the nonpregnant or in men, in the absence of other conditions. Brooker reports that enamel is not destroyed until the acid concentration is 100 times that of normal saliva.

Blue litmus turns red when in contact with carious dentin, but litmus changes at a p_H of 7.0 and will turn red any time if placed in contact with gingival tissue.

The Jones-Larson report, in which many teeth were decayed before eruption, would place the teeth entirely beyond the reach of local acid changes and demand the presence of a systemic condition, which must of necessity be an acidosis or some form of physiologic unbalance such as a calcium deficiency or an inactivity of the alkaline neutralizing or immunizing agent.

In this connection, it is not amiss to add a word on mouth washes. Hanke declares that the most scrupulous care of the oral cavity is no insurance against decay. Bunting's report concerns three groups of children who had a diet which included an abundance of milk, fruit and green vegetables and was otherwise well balanced. In these groups, caries was diminished or entirely prevented whether aseptic mouth washes were or were not employed. In group diets which varied from the normal standards, caries was not arrested, but continued active regardless of the presence or absence of aseptic lotions.

Bunting's report brings us to the consideration of the highly important factor of diet in caries. Research into dietary conditions has established beyond question that food has an inherent and fundamental relationship to the occurrence of caries.

Boyd and Drain had diabetic children placed on a diabetic diet with cod liver oil and no cereal. The active carious areas hardened and the process ceased. Normal children were placed on a cereal-free diet and the carious pits healed and the process stopped in ten weeks. The inference is unescapable that the exclusion of cereals hastens the cure of caries.

In Mellanby's cases, again, the cereal was not removed from the diet, but in one group oatmeal was added, and in this instance the occurrence of caries was more frequent. Mellanby therefore concludes that cereals interfere with calcification. In another series, she used two diets consisting of (a) mostly milk and meat, eggs and cod liver oil, and (b) less milk, and eggs and no cod liver oil, but also oatmeal. She found, after eight months, that caries occurred four times as often on the second diet as on the first.

Perhaps vitamin D should be added to that in the cod liver oil. Vitamin D in the form of viosterol facilitates the absorption and utilization of calcium, while the deprivation of vitamin A, found in cod liver oil and elsewhere, favors focal infection. Hence, an abundance of these agents must be provided to protect the teeth and gums in pregnancy.

Hawkins declares that the acid base balance and calcium-phosphorus balance with vitamin D are the principal factors in caries. In his opinion also too high a percentage of cereal foods is a mistake. Cereals should not exceed 20 per cent of the diet and, when necessary, the diet should be corrected with vegetables, meats, fresh eggs and cheese, with the addition of vitamin D in the form of cod liver oil or viosterol.

Apparently, even a high blood calcium may not be utilized owing to defects of

metabolism through lack of vitamin D. Sherman, Davis, Boyd, Drain and Nelson, Bunting, Hadley, Jay and Hard, Richardson and Hanke believe that dental caries can be arrested if the patient will diet properly and Mellanby's striking experiments confirm their opinion. The daily minimum requires a pint of orange juice, one lemon, tomatoes, eggs, calcium lactate and vitamin D, either as haliver oil, cod liver oil or viosterol. Jones and Larsen further report, in their series, on two breast-fed babies whose badly decayed teeth erupted at the ninth month. After six months of free diet on cow's milk, fruits and vegetables, etc., the decay was arrested.

From the data thus far presented, it does not seem unwarranted to assume that dental caries is due primarily to a deficiency in the blood of calcium or phosphorus or both, or to an inadequate utilization thereof owing to the absence of vitamin D, which leaves the teeth unprotected.

Brooker states that the chemicoparasitic theory of caries held by dentists is almost a complete explanation. The difficulty rests in the word "almost." This hypothesis does not adequately explain the occurrence of caries, with or without plaques, unless a deficiency of the alkaline buffer in the blood and saliva is accepted. It certainly does not explain caries in unerupted teeth.

With a deficiency of calcium or phosphorus or both, the chemical accessories in the mouth may become effective and possibly satisfy all the conditions not alone for the pregnant but for the nonpregnant and for men. That immunity to caries is uncommon in children and inconstant in adults, especially in pregnancy, is not inconsistent with the theory of protective mineralization which the data herewith presented would appear strongly to support.

Therefore, the statement may be repeated that, in consequence of defects in mineralization through absence of vitamin D, dental caries ensues. It is immaterial to the purposes of this paper whether the mineralization is found to be deficient in the blood, the salivary secretion or both.

If the study now moves to inflammation of the gums, a slightly different pathologic process is revealed.

Rickets at present is recognized as a calcium deficiency disease, with a lack of phosphorus also and in particular of vitamin D. Vitamin D, the "antirachitic factor," the "calcifying factor," is a substance essential to normal calcium metabolism and brings about increased retention of calcium. (Cantarow.) At present, it is administered to best advantage as viosterol.

Scurvy is often associated with rickets; and probably vitamin C, which is the vitamin lacking in scurvy, is also occasionally lacking in rickets. Both rickets and scurvy result from calcium and phosphorus deficiency, not necessarily from diminished blood content, but from defective utilization on account of the low phosphatic concentration in the blood and especially from lack of those dominant vitamins C and D. Calcium and phosphorus metabolism are very intimately related.

Scurvy is one of the oldest diseases known. In addition to the more or less obvious hemorrhages which occur from the gums and elsewhere as the disease progresses, there is a failure of the normal deposition of calcium and phosphorus in the bones, though the diet may contain an abundance of both elements.

Salter has recently made a study of scurvy to determine whether the stores of lime salts in the bones were depleted while the calcification of the growing

portions were inhibited through lack of the antiscorbutic vitamin, C.

At various stages of the experimental disease, the missing vitamin C was supplied as orange juice and, at the same time, alizarin was injected so that the newly deposited bone salts would be stained red and easily detected. The Boston investigator discovered that, in healing scurvy, the epiphyseal line was especially well marked and showed a deposition of the salts in the zone of rapid calcification.

Furthermore, the cancellous tissue of the epiphysis and diaphysis was stained and the pink trabeculae extended a small distance into the medullary cavity. The shaft too was colored by the dye distad from the epiphysis, but not so deeply as the trabeculae. On the contrary, the bones of animals receiving no vitamin C were not stained by the dye, though their ration contained calcium and phosphorus.

As in rickets and dental caries, then, defective calcification of the bones is one of the signs of scurvy. The demonstration that this disease affects the storage of bone salts in the trabecular areas as well as their deposition in the epiphyseal tissue is important, since it shows that the increase and functional activity of bone cells can be encouraged by an abundance of antiscorbutic vitamin.

Dalldorf has established the fact that the hemorrhagic diathesis in experimental scurvy develops earlier than any other known sign of the disease and persists in some degree throughout. Gingivitis in pregnancy is a typical illustration of this phenomenon.

Scurvy is essentially a capillary disease. The pathologic change in experimental scurvy (Findlay) is swelling and degeneration of capillary endothelium whereby the flow of blood through the capillaries is retarded and congestion ensues. The degeneration of the endothelium damages the intercellular substance and edema and hemorrhage follow, while stagnation in the blood stream causes deficient oxygenation of the tissues.

Hanke finds experimentally that a diet rich in vitamin C will not infrequently reduce the thickness of the peridental membrane and change soft, spongy hyperemic gingival tissue to normal tissue.

The pulp, the peridental membrane and the gingival tissue are histologically continuous and have a common embryologic origin. It is probable therefore, continues Hanke, that pulp changes in the human subject may occur even earlier than corresponding alterations in peridental and gingival tissue.

The evidence all leads to the conclusion that deficiency of vitamin C is an important factor in producing changes in the pulp, the peridental membrane and gingival tissues. If this is true, vitamin C deficiency goes still farther to controvert the theory that dental caries, though a chemical process, is purely local and due to bacterial fermentation, as well as to deny that sponginess of the gingival tissue and pyorrhea are due to bacteria which cannot be removed from the mouth. Cellular metabolism is the main issue. If healthy cells can be produced by appropriate nourishment, it might be possible sometimes to eliminate bacteria from infected areas and keep them out.

This part of the subject has been discussed with some tediousness, not only for its intrinsic importance but also because it plays so significant a rôle in my thesis, the elaboration of which leads to a consideration of the logical extension of the calcium problem to its influence in eclampsia.

Guanidine, according to Major, is toxic to both muscle and liver tissue. This observer maintains that the presence of guanidine is responsible for eclamptic

convulsions and also that it is the toxic factor in uremia, osteomalacia, liver necrosis and the tetany of pregnancy which Richardson has emphasized. This agent is a waste product of voluntary muscle action which, like the acidity of the mouth, is neutralized under normal conditions by calcium.

Mitchell first called attention to the value of calcium in eclampsia. Drennan, Kehrer and others exploited the idea. Practically, we learn that prompt relief of the symptoms has followed the intravenous injection of 10 c.c. of a 10 per cent solution of calcium gluconate in eclamptic and preeclamptic states. The subsidence of the serious features of the attack may occur as quickly as in twenty minutes after medication. Lopez has had good results from injections of 20 to 30 units of parathyroid hormone.

The strict milk diet, which has been almost routine in the therapy of this disorder, can be evaluated now, not on the ground that it excludes other and more toxic foods, but because it furnishes the necessary calcium in an easily assimilable form.

Minot and Cutler, quoted by Cantarow, noting the resemblance between the pictures of eclampsia and acute hepatic injury in dogs, studied the guanidine content of the blood in eight patients with preeclamptic toxemia and four with eclampsia. Guanidine was increased in every case. Striking relief from the urgent symptoms followed the intavenous injection of calcium gluconate in both types. In a few hours, the convulsions ceased, the headache and dizziness disappeared and the blood pressure dropped. These experiments have been reproduced in the prenatal clinic at the Northwestern University Medical School by Drs. Richardson, Gregory and Serbin, with similar success. Thus, a definite group of disorders confront the doctor or dentist. What can he do to control them?

The treatment of these phases of hypocalcemia follows easily from the etiology. Vitamin C is abundant in orange juice and milk. Vitamin D is readily available in cod liver oil, haliver oil and viosterol (irradiated ergosterol) or even in the normal skin when exposed to sunlight or the mercury lamp. Viosterol is the most satisfactory source of the vitamin because it not only increases absorption from the gastro-intestinal tract but also encourages the storage of calcium and phosphorus in the spongiosa of the bones. Indeed, calcium in the absence of vitamin D, just as phosphorus in the absence of vitamin C, is neither absorbed nor utilized. Mellanby's experiments with diets a and b clearly show the value of cod liver oil when teeth are breaking down.

These numerous investigations and diverse experiments all led us to conclude that dental caries, whether associated with pregnancy or not, is due to dietary conditions wherein a deficiency of vitamin D and calcium is the most conspicuous factor. It seems equally plausible to conclude that the gingivitis of pregnancy is due to a deficiency of calcium and phosphorus, together with a lack of vitamins C and D, and that this particular complication falls regularly into line as a premonitory symptom of scurvy.

The evidence also seems to indicate rather clearly that dental caries and gingivitis, which lead to tetany, rickets, scurvy, osteomalacia and possibly to eclampsia and acute yellow atrophy of the liver, form a common group of calcium deficiency disorders with phosphatic complications as important accessories.

The therapy, then, must be largely dietary. For instance, the premonitory symptoms of tetany such as fragility and brittleness of the teeth, caries, defects of the enamel, hyperemia of the pulp, brittle-

ness of the hair, longings, loss of the nails, cachexia, muscular weakness and, above all, the nocturnal cramps in the legs, tachycardia and convulsions, which Richardson has so adequately described, may be quickly relieved by adding to the diet an excess of milk, cream, eggs, butter, fruits and vegetables, with a low cereal selection and supplying also calcium lactate or chloride, viosterol or cod liver oil with sunshine or the rays of the ultraviolet lamp. The Mellanby reports also strongly accent the importance of vitamin D, found in haliver and cod liver oil and viosterol, and state that ordinary carious processes are arrested sometimes in two weeks; that no new cavities form, and that the carious surfaces show increased hardness from the recalcification of the disintegrated dentin.

In gingivitis and pyorrhea with redness, swelling and bleeding of the gums and loosening of the teeth, the cereals must be omitted and a diet prescribed of raw whole milk, buttermilk, oranges, peas, beans, cabbages, tomatoes, etc., for vitamin C, and for vitamin D, as previously indicated, viosterol.

According to Toverud's experiments, a positive and highly needed mineral balance can be assured to the pregnant or lactating woman if she adds to her dietary regimen a pint or more of milk daily, with fresh fruits, eggs, vegetables and viosterol.

The Boyd and Drain and the Mellanby reports on cereals constitute a contribution to infant feeding and adult nutrition which must not be overlooked.

Incidentally, it would appear not only that bacteria cannot be the destructive factor in caries, but also that mouth washes, antiseptics, powders, tooth pastes and similar agents have no value beyond the temporary comfort conferred, and carry with them a deceptive sense of security, since the mouth and gums cannot under any circumstances be made germ free during life.

Finally, we must conclude, as Cantarow says in his introduction, "that the subject of calcium metabolism occupies a position in current medical literature comparable to that of carbohydrate metabolism of some years ago."

30 North Michigan Avenue.

PERIODONTIA: ITS RELATION TO THE ECONOMICS OF A COMMUNITY IN ITS SOCIAL ACTIVITIES*

By HENRY E. GERMANN, D.D.S., F.A.C.D., Cincinnati, Ohio

PROGRESSIVE dentists are cognizant of the economic value of dental service, especially prophylaxis service, to both individuals and the community. We have carefully studied all phases of economics from prevention to cure, in both private and social welfare practice, but what have we done to disseminate this knowledge? We have given much of our time to the education of patients, of children and mothers through the schools, of members of all types of organizations and of workmen in the factories. Thousands of dollars

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have been spent in the education of the public by means of paid publicity in periodicals and papers, by radio and by many other types of educational publicity, in an effort to emphasize the importance of a clean and healthy mouth, and its influence in the maintenance of a healthy body.

Within the present year, there has been much discussion regarding the program of publicity as adopted at the American Dental Association meeting at Memphis. It is not my desire to reopen the discussion of this matter, as to its good and bad features, but I will make this positive statement: To every individual, be he rich or poor, who has been influenced by our efforts at education, and is convinced that dental and prophylaxis service are necessary to the maintenance of good health, we must supply a service commensurate with his earning power and ability to pay. This is a challenge and a moral obligation which we cannot and dare not evade. If we do not assume this responsibility, we shall be accused of advertising for personal gain alone. It probably is within the province of the tradesman, the man who sells tooth pastes, toothbrushes, etc., to say "See your dentist twice a year," without worrying about whether every individual who hears him has the money to do this. But when we tell the public the same thing, and those who have a very limited income and many more of large family who have an income insufficient to keep body and soul together, say to us, "Where shall we go for our dental service?" we find ourselves in a very embarrassing position. At present, we cannot supply this service, and if we do not interest ourselves in the situation to the fullest degree, we shall naturally be adjudged by the public to be advertisers for our own special gain.

Our education program should be subdivided into three phases: 1. Education of the public on the subject of mouth hygiene. 2. Education of the dental profession as to its definite moral obligation to the public. 3. Education of the taxpayer as to the economic value of health conservation to a community.

It is important that the public be educated as to what constitutes a healthful and necessary service. They should also be made cognizant of the evils of some types of service received in certain dental parlors, but this cannot be done until we have organized and put into operation clinics which will supply to all classes of people seeking dental health a service which will be preferable to dental parlor service. The education of the public is fairly well organized. That it has made progress is evinced by the fact that multitudes of people would seek dental service, in order to improve and maintain their health, if there were places where they could receive such service as we recommend at a price they could afford to pay.

Along with education of the public comes the task of educating the dentist. Many there are who are not fond of work and who are simply looking for an easy time; too many who cannot see the handwriting on the wall; too many who are unwilling to give freely of their unoccupied time to intelligent unfortunates, many of whom will not always occupy their present social status. No doubt many of these charity or semicharity patients will, after becoming conscious of the importance of dental care, develop into good patients for some dentist.

In making this assertion, I am reminded that a tremendous amount of education will be necessary to lead all of the members of the dental profession into this line of thought. Nevertheless, I am convinced that if the profession does not assume the responsibility of

planning and promoting a dental service for all classes of people who want it, others less interested in the profession and less capable than we are will do it for us.

The education of the taxpayer becomes a more difficult problem as well as a most important one, for the future of dentistry as a dignified profession depends on our success in establishing a definite program to care for all classes of people.

The expense of such a dental program must be borne by the taxpayer, and not by the dental profession; and since the average taxpayer is not socially minded, his conversion must be brought about by a campaign on the economic value of a dental service to a community. We must talk to him in the language of dollars and cents.

Up to the present time, the layman has accepted this education and quite naturally has interpreted it to be for himself and, if there be dependents, for them also, this being about as far as it has gone unless he was interested in social service work. This is quite natural, since we have not given the economic value of dental service to a community the proper study and publicity. The taxpayer must be taught that every neglected mouth may at some future time become a liability of that community, a taxpayer's liability. He must be shown the value of preventive dental service, and how an expenditure of funds in the right direction will greatly reduce diseases which are the result of neglect of oral conditions, thereby reducing the number of patients, finally drifting into the hospital and other health institutions to become the taxpayer's burden. In most of the European countries, the wage earner is recognized as an important person, and every effort is made to supply him with health service, this being done by various types of insurance. In Russia, most of the dentists are employed by the government to serve the working classes only.

Private dentists in Russia have a rather hard time, finding it difficult to rent sufficiently large quarters and especially hard to buy instruments and supplies, as the government takes what it needs first, the remainder being available only at exorbitant prices.

You will say, "Nothing like that will ever happen in this wonderful country of ours." True, it may not, but do not overlook the fact that almost anything can happen in this land of politicians, as has been proved to us in the last few years.

In making an analysis of a city like Cincinnati, with a population of 450,000, we find the following condition: We support a general hospital housing 839 patients daily at an annual cost of $897,000, not including the investment in grounds, buildings and equipment. Of course, it would be rather difficult to estimate just how much of this expenditure could be prevented with adequate preventive treatment for these patients in early life.

In the Cincinnati Schools, it costs an average of $123.23 to educate each child in an intermediate grade; 3,756 failures last year in these grades meant an expenditure of about $465,744. It costs an average of $170.75 to educate each child in a high school; 2,313 failures in this group last year cost the taxpayer about $394,944, or a total of about $860,688 to reeducate children who failed to pass their respective grades. Think of it! Almost a million dollars to reeducate children each year. How much health service would this buy for children in the school? And this is not the end of the story, because many of these neglected children become wards of the taxpayer and philanthropist sooner or later,

because of their handicap. Because of disease which has been neglected in childhood, they do not grow up to be strong men and women, with the result that our general hospital, infirmaries, public health institutions and community chest are all taxed to capacity, and, at this time, we are building more to take care of them. Of course, it is impossible to estimate exactly the number of children who fail in school because of bad teeth and unhealthy mouths and disease which is the result of these oral conditions. Authorities vary in their estimate from 15 to 30 per cent, reeducation costing from $129,000 to $258,000 a year, more than a quarter of a million dollars. Add to this only 15 per cent of the hospital expenditure, $135,000, and we have a sum amounting to $385,000 expended as the result of neglect of oral conditions, the most of which could be saved with an annual expenditure of one-third of that amount. Think of this expenditure of $385,000 traceable to oral neglect as against $30,000 of taxpayers' money spent at the present time for preventive dental work. With figures such as these, we can show how much preventive work could be accomplished with only a portion of this outlay of money. With the great number of taxpayers averse to the expenditure of funds for the alleviation of sickness and suffering, we must stress the fact that preventive dentistry alone will be a big factor in the saving of funds.

We could conscientiously paint a much blacker picture of the expenditure of funds for the unavoidable sickness in a community, and I am also satisfied that a much large number than is represented by 15 per cent are supported by taxation in the hospitals and other institutions, because of neglected mouths. So you will see that by making an analysis of any community along these lines, we have the opportunity of convincing the taxpayer, even though he is not socially minded, that, by the expenditure of one dollar in the preventive field, four or five can be saved in the corrective field, and we all know that this is the only way in which we shall win over the taxpayer.

Most health boards and school boards would like to do this preventive work, but up to the present time the only pressure which has been brought to bear on them and the problem has come from the dental profession and social worker and not from the taxpayer, and it is the backing of the latter that we need.

There is a increasingly great effort being made all over the country to broaden dental service for the deserving classes. Various plans have been adopted in different localities and good work has been done. A careful study and analysis of these plans by the American Dental Association, with reports and advice to every component affiliated with the Association would be very helpful to the profession in carrying out this plan of education, which I am sure will have a greater influence with the taxpayer's support of our problem than all possible newspaper, magazine and radio publicity.

In Cincinnati men have volunteered to work from two to six hours a week in their individual offices for high school students unable to pay. Many more are giving up one-half day a week in the outpatient departments of the hospitals. I am sure that any of us would prefer to give a child who is starting out in the world dental service rather than to sit around worrying about the lack of business or be out fishing or golfing. A summary of this work from June 1 to September 1 proves that our dentists are not wholeheartedly interested in the situation. This is proved by the ratio of time volunteered to that given:

145 dentists have accepted 414 cases; twenty of these 145 dentists have taken

122 of the 414 cases; 13 per cent of the volunteers have done 30 per cent of the work; the time volunteered in this three months' period was approximately 5,220 hours; the time actually given was 532 hours, about 10 per cent of that promised.

Many dental societies have adopted various plans to care for the indigent, but volunteer service in the office of the dentist cannot be satisfactorily carried on.

Last month's issue of *Oral Hygiene* contains a four months' report of a clinic at Pittsburgh, as follows: 1,266 patients taken care of in four months, the service including 1,328 extractions and 1,272 fillings:

1. A committee of three was appointed to promote and manage the clinic.

2. One hundred and fifty members donate their services. Each operator serves a half-day at a time, two being on duty in the morning and two in the afternoon.

3. The local charity organization donates the services of a nurse and an attendant.

4. The clinic was furnished in the following manner:

(a) The leading professional building donated three offices.

(b) Local dental supply houses loaned the complete equipment.

(c) The local telephone service is given at the charity discount rate and the Child Health Council takes care of the bill.

(d) A local printing concern donated admission and record cards.

(e) The building donated the plumbing fixtures and service.

(f) A local towel supply house launders the towels, which were donated by members.

(g) Anesthetic gases were donated and two of the local exodontists care for difficult cases in their own offices.

(h) Dentifrice concerns donated toothpaste and the oral hygiene committee of the society provided toothbrushes.

(i) Stationery, signs, etc., were donated.

(j) Dental materials are provided at cost price.

(k) Local newspapers were generous in giving publicity and school authorities have cooperated in investigating needy cases and bringing children to clinic.

A great deal of good has been done by this clinic in the few months that it has operated, and this without working a hardship on any one person or concern. The society regards the clinic as a temporary emergency effort during present day conditions.

The needy must be cared for, and if organized dentistry does not care for them, we can look for outside and perhaps unwelcome intervention. These programs when adopted create a good feeling in a community. Many of us have pleasure in doing something worth while for some one else. There are others, and good dentists, who say that we are fools. I shall not criticize any one for his attitude. We feel that the above mentioned method of handling this work is entirely inadequate and incomplete and is merely a palliative for an existing condition.

If we are to protect the best interests of the profession, we must put our shoulders to the wheel and carry out a program of education. There must be no slackers in this effort to establish a permanent plan for caring for all classes of patients. One of the weakest links in the whole chain of social service work at present is the social service rating of patients by the committees that investigate the social standing of applicants; and until sufficient funds are appropriated to make careful checkups regarding the statement of the applicants as to their

financial status, dentists will refuse to give of any of their time and capital, and taxpayers will not support any program.

Every one of us has had the experience of hearing complaints from Mrs. A. that because her child states the truth fully in school that he has a dentist, she is compelled to go to her own dentist and pay for service, while Mrs. B.'s child, who states that he has no dentist, receives gratuitous service in the school prophylaxis clinic, and that since Mrs. B. has much more money than Mrs. A., this is not fair. Much time could be spent in citing many other types of irregularities which tend to break down the morale of any service movement and can be corrected only by adequate social service investigation.

I therefore strongly advocate the establishment of a social service investigation bureau.

Summarizing this subject of economics we find that (1) a program of education for the layman; (2) coordination of effort on the part of all dentists; (3) support on the part of school boards and boards of health; (4) education of the taxpayer as to the economic values involved and the need for his cooperation; (5) establishment by the community and under the profession of free clinics for the indigent and pay clinics within the range of all classes and (6) thorough investigation of all applicants for indigent or part-pay clinic service, as to their ability to pay, are necessary factors in the protection of this important and necessary branch of the medical sciences (dentistry), *our professions.*

VULCANITE VS. SUBSTITUTES*

By E. B. OWEN, D.D.S., St. Louis, Mo.

WERE there no need for a substitute for vulcanite, there would not be so much effort to supply one.

Although vulcanite has served us faithfully for eighty years, we all realize its shortcomings, chief of which is its unsatisfactory color; but it must be borne in mind that a substance intended to displace vulcanite must possess other properties than a pleasing color.

For many years, some effort has been made to find a more suitable plastic material for denture bases than vulcanite, but only in the past six years has there been such determined effort, and, during this period, many materials have been offered. Competition and lack of funds on the part of the manufacturer, and eagerness on the part of the dentist, have been the cause of many improprieties on the part of both. Manufacturers have sponsored, advocated and untruthfully advertised materials that were untried, unproved and unfit for use in the mouth. These products were sold at high prices to the dentist. The dentist's patients held him responsible, and he, with his usual conscientiousness, assumed the responsibility, and consequently was, or will be, the loser. Thus, the dentist has paid the bill for whatever development may have been made in these materials, with-

*Read before the Odontographic Society of Chicago, Oct. 3, 1932.

out a chance of sharing in the enormous profits.

Some of these materials, especially the celluloids, have been sold to the dentist at such profit that one manufacturing concern is said to have amassed an immense fortune in the few years they have been in business.

Many of our dental journals have carried advertisements proclaiming these materials as possessing strength, form retention, nonporosity, enduring beauty and various other properties that they do not possess. It is wrong that manufacturers should misrepresent their goods; it is wrong that dental journals should accept such advertising matter. It is all misleading to the dentist. But the dentist himself, I must say, is not entirely blameless. If he accepts blindly everything he hears and reads, he has no one to blame but himself. Unfortunately, the dentist accepts everything the sponsor of a material says about it, and investigates nothing.

It is surprising that the American Dental Association committee investigating materials has not made a report on the various plastic denture base materials. Perhaps lack of time is the only reason.

The dentist, having gained his information from advertisements, proceeds to pass this information on to the public. Dentures are exhibited and patients are advised of the good qualities they possess in addition to beauty. "You can throw them across the room and stand on them without breaking them. Higher priced,

Relative Merits of Various Plastics*

Vulcanite	Properties	Condensites	Celluloids	Glycerine-Polybasic Compounds
B	1. Strength	C	A	B
A	2. Permanency of form	A	C	A
B	3. Absence of absorption	A	C	A
B	4. Tissue tolerance	A	B	A
A	5. Length of life	C	C	?
B	6. Objectionable taste and odor (lack of)	B	C	B
C	7. Pleasing color	B	B	B
B	8. Permanency of color	C	C	B
B	9. Expansion and contraction in curing (absence of)	A	A	C
B	10. Ease of repair	C	C	C
A	11. Time of curing	A	A	C
C	12. Retention by porcelain and metals	C	C	A

*A, good; B, fair; C, poor

of course, but worth it." Propaganda like this has been spread until no one now is satisfied with a vulcanite denture. When we tell the patient the truth concerning the drawbacks of the other materials, he thinks that we are trying to avoid a responsibility that is justly ours, or trying to persuade him to take a cheaper material. The one question in the mind of a patient who is to wear dentures is "What kind of material do you use?" If dentists had spent as much time emphasizing the value of dentures as a health service as they have talking about different materials, dentistry would have a better professional standing.

When patients come to me for dentures, they are given the facts as I know them. They are informed that the purpose of dentures is to afford them the best possible substitute for their natural teeth; that they may masticate their food and thereby promote and preserve health; that their facial expression may be restored and preserved and the teeth be so constructed as to preserve the alveolar ridges in order that they may wear artificial dentures comfortably for a greater length of time. All through the conversation, service, health, comfort and appearance are emphasized. The subject of materials is kept in the background, although it must, of course, enter into the conversation. The patient will bring up the subject. As a matter of secondary importance, they are told they may have any of the plastics they desire at the same fee, as the fee charged is solely for service and no charge is made for materials. I emphasize the fact that the best materials we can buy cost very little, and, even for metal bases, the higher fee is for extra time and skill required to manipulate the metal and not for the materials themselves. If we emphasize the cost of material, the patient thinks that the difference between the small cost of material and the large price of a denture is too much profit; but, if our fee is for service and nothing is charged for materials, they have no way of estimating the cost of that service. They consider the charges as a fee and not as a price.

Patients are told what they may expect from vulcanite and from the condensites and celluloids, and thus they are in a position to choose a material. They almost invariably ask what I would recommend, and, in most instances, I recommend vulcanite; but not until the patient is fully informed regarding the materials available, and usually when the selection is made, it is his selection and his responsibility.

My observation and experience with the vulcanite substitutes has established, with a fair degree of accuracy, the fact that the range of life of dentures made of these materials is from one year as a minimum, to five years as an extreme maximum. The condensites will retain their form and last until they break, which may be twenty-four hours or an indefinite period, but the color, which is the only excuse for their existence, cannot be depended on for more than one year, two years at the most. The celluloids begin turning brown usually within one year, sometimes much sooner, and, at the end of five years, the pins are usually exposed and the material pulled away from the necks of the teeth. With these facts all before him, and the likelihood of having to have new dentures within a few years, if the patient thinks it worth while, for the sake of appearance, to accept the substitutes, all well and good. At least, he cannot accuse me of deceiving him as to the properties of the materials.

The relative merits of the various plastics may be better understood by a comparative table which I have prepared. Listed therein are the properties that a successful denture base must possess, and

the degree of conformity of the various materials to these requirements is indicated by the markings. In the table, vulcanite, which is the standard with which all other materials must be compared is on the left. The substitutes are on the right. Among the substitutes is found the glycerine polybasic group, or bakelites. These contain, in addition to the ingredients of condensites, glycerol and certain fillers. Only one of this group, bakelite C 25, is known commercially. It is sponsored by the Bakelite Corporation of America, and its use is not being encouraged until certain properties are improved on; a very commendable attitude. My experience with this group has been with another material not generally known to the dental profession as yet.

If we should strike an average of the various materials, we would find that there is not much difference, but it is not possible to strike an average from a practical standpoint because of the unequal importance of the various requirements, the first six being far more important, in my estimation, than the last six. A grade of C or "poor," in any one of the first six requirements, in my opinion, disqualifies the material. Seven and eight are highly desirable but not absolutely essential. Vulcanite grades C in property 7, yet vulcanite has made acceptable and serviceable dentures for years.

CONDENSITES

The phenol-resins, or condensites, are composed of phenol and formaldehyde condensed in the presence of heat from the liquid to the semisolid state, in which they are marketed, and afterwards, again in the presence of heat, are cured into a rigid and more or less brittle and infusible state. These materials, once cured, retain their form and are quite dense, permitting but little absorption. They grade high in tissue tolerance and pleasing color, but, unfortunately, low in strength and permanence of color. From my observations, the color remains good for not more than one year. The strength of these materials has been improved, and when considerable bulk may be used in the denture, it is fairly safe to employ them, but the color, or rather the permanency of color, apparently has not been improved. On the other hand, announced improvements in some cases have proved to be retrogressions.

CELLULOIDS

The earliest substitute for vulcanite was offered in the form of a nitrate of cellulose under the name of parksine, taking its name from Dr. Parkes who presented it. This was as early, perhaps, as 1860. A few years later, the brothers Hyatt, of Newark, N. J., improved on parksine and called the product celluloid, getting a patent or copyright on the name. Celluolid is composed of nitrate of cellulose, camphor and coloring. It became quite popular as a denture material, but, thirty or forty years ago, it passed almost entirely out of use, and was forgotten in the field of denture construction. About fifteen years ago, it was reintroduced, this time under the name of mother gum. It failed to impress the dental profession. During the World War, a celluloid known as hecolite was brought out in Germany as a substitute for rubber, of which there was a great shortage. Its color appealed to Americans and the right to manufacture hecolite was soon transferred to this country. It became the pioneer in the present-day widespread use of celluloids. It was soon followed in the American field by alcolite, agalyn, parfait and many others. There is some little variation in the manufacture of these materials, but basically they are nitrate of cellulose plus camphor (except in one instance where a substi-

tute for camphor is used) and must be considered as celluloids. They have some properties that recommend them, such as tremendous strength and a color that is an improvement on that of vulcanite. The disadvantages are numerous: high percentage of absorption, deterioration with age and instability of both form and color. As I have been able to observe the behavior of celluloids, the change in form, or warpage, is due to two distinct conditions: The first I call, for the want of a better name, the "green stick tendency." When we bend a green stick and release the force, the stick gradually straightens out again. When we change the shape of a block of celluloid under pressure, there is a tendency, when the pressure is released, for it to resume its original shape. This property is not true elasticity, but rather a molecular tension, with the molecules tending to assume their normal arrangement when the tension is released. In the case of artificial dentures, this molecular tension manifests itself almost entirely within the first forty-eight hours. It is not an excessive change and apparently becomes less in ratio to the length of time the material is kept under pressure.

The second and most important change in form is due to expansion and contraction. The material, being porous and cellular, absorbs a large amount of moisture and the cells expand in the presence of this moisture. When the material dries out again, it contracts. An interesting experiment to illustrate this property can be made by running a nonexpanding stone cast into a dry celluloid plate, soaking the plate several days in water, then running another cast into the soaked plate, alternately drying out and soaking the plate and trying it on the two casts. The dry plate will be too small for the cast run into the wet plate, while the soaked plate will be too large for the other cast. An attempt to control this property of the material by using a strong gold brace in the palate of the denture met with considerable, but not complete, success. In view of this property, I recommend the wet method of pressing dentures that are intended to be worn in the mouth, in preference to the dry method used by most of the laboratories.

GLYCERINE-POLYBASIC COMPOUNDS

The glycerine-polybasic group of substitutes are composed, as far as I know, of the same materials as the condensites, plus glycerol and certain fillers to produce strength. Little is known scientifically of this group, as the formulas and patents are controlled by commercial institutions. We have some practical knowledge of them. They are known in the commercial field as bakelite and are used for pencil barrels, insulating materials, radios, etc.

Like the condensites, they are cured by the application of heat and pressure and pass from the fusible to the infusible state, undergoing complete chemical change. While this curing process for the condensites is a matter of only forty minutes, for the gylcerine group it requires about sixty hours, all told, including ten or twelve hours for drying out the mold. This long curing time is a decided disadvantage, but, from experience and observation thus far, the finished product appears to be decidedly superior to the condensites. They are apparently as strong as vulcanite, are dense and therefore practically free from absorption. They retain their form absolutely, and adhere tenaciously to porcelain and metal, a property that would prevent pulling away from the necks of the teeth. The color, after the outer surface has been polished off, is very pleasing, but my experience with it does not cover enough time to determine whether the color is permanent. It seems to be over a period of eighteen months.

These materials, of which there are only two to my knowledge, are not released for general use by the profession because of two shortcomings: the long curing time (sixty hours) and an excessive expansion and contraction in curing faults which the manufacturers hope to correct before introducing these materials to the profession. They hope also to prove permanency of color over a period of years. This attitude on the part of the manufacturers is highly commendable.

SUMMARY

1. Vulcanite has its faults, but it has served the profession faithfully for eighty years and has not yet met its Waterloo.

2. Condensites are not dependable, but they have their uses. They are acceptable where large bulk may be used, but full consideration must be given to the fact that the color is not permanent.

3. Celluloids are the most widely used and perhaps the least desirable of the materials, and, like the condensites, should be used only after a thorough understanding with the patient as to their objectionable properties.

4. It is believed that vulcanite, except for occasional use, together with the condensites and celluloids, will eventually be displaced by some material in the polybasic group and that the time when this will take place is not far distant.

(Ed. note.—After this article was in type, the author requested the privilege of amplifying it in a subsequent paper in order to bring the subject up to date in accordance with the latest findings. A second paper will therefore appear in a future issue.

ADDITIONAL LIGHT ON THE ETIOLOGY AND NUTRITIONAL CONTROL OF DENTAL CARIES WITH ITS APPLICATION TO EACH DISTRICT SHOWING IMMUNITY AND SUSCEPTIBILITY*

By WESTON A. PRICE, D.D.S., M.S., F.A.C.D., Cleveland, Ohio

AN experimental basis for a new theory regarding the phenomena of dental caries has recently been presented.[1] The data presented provide an explanation of many conflicts in interpretations made from available clinical and experimental studies and provide a bridge between the two main opposing views. The first, growing out of the work of Miller, Black and Williams, placed the responsibility on the action of bacteria as a controlling factor in the environment of the tooth and, hence, on the need for oral hygiene. The second placed the responsibility on metabolic processes, and hence was interpreted to be largely of nutritional origin. This theory has been supported by the work

*Read before the Section on Histology, Physiology, Pathology, Bacteriology and Chemistry (Research) at the Seventy-Fourth Annual Session of the American Dental Association, Buffalo, N. Y., Sept. 15, 1932.

1. Price, W. A.: New Light on Etiology and Control of Dental Caries; J. D. Res., abstr. 12:540 (June) 1932. Experimental Basis for New Theory of Dental Caries with Chemical Procedures for Determining Immunity and Susceptibility, D. Cosmos, 74:1139 (Dec.) 1932.

of McCollum, Mellanby, Price and others. A harmonization of these two theories is needed for practical progress in the control of dental caries, which is apparently on the increase in most of the civilized world.

The experimental and clinical data reported in the two communications referred to above can be summarized as follows: The routine chemical analyses of the blood from 400 successive cases revealed that when powdered bone chips were placed in blood serum or plasma, there was, in normal cases, a movement of calcium and inorganic phosphorus from the blood serum to the bone chips, but that, in certain pathologic conditions, frequently the movement of inorganic phosphorus and occasionally that of calcium was in the opposite direction; namely, from the bone chips to the blood serum or plasma. For sixty successive cases, it was found that, in fifty without marked evidence of decalcification of the skeleton, there was a reduction of 27 per cent in the calcium of the serum and 18 per cent in the inorganic phosphorus. In the ten others of this group, all of which were characterized by marked decalcification, the calcium moved from the blood serum to the bone chips in an amount of 24 per cent of the serum content, while the inorganic phosphorus moved in the opposite direction; namely, from the bone chips to the blood serum in an amount equal to 28 per cent of the inorganic phosphorus content of the serum. Studies were made on the effect of treatment through reinforcement of the nutrition with high mineral carrying foods and fat-soluble activators, the latter as provided in exceptionally high vitamin butter and high vitamin cod liver oil. In two of the patients of the decalcifying group, one presented spontaneous fracture in which, before treatment, the inorganic phosphorus moved from the bone into the serum in the amount of 35 per cent of the total, and after treatment it moved in the other or normal direction in the amount of 15 per cent. Roentgenographically, the bones showed an increased density, with progressive calcification. In the other case, one of degenerative arthritis with marked decalcification in which, before treatment, the inorganic phosphorus moved from the bone chips to the serum in an amount of 11 per cent, after treatment with nutritional reinforcement with high mineral carrying foods and fat-soluble activators (the latter as provided in exceptionally high vitamin butter and high vitamin cod liver oil), the inorganic phosphorus now moved in the other or normal direction in the amount of 34 per cent.

Since dental caries presents so much evidence of being influenced by the environment of the tooth, large numbers of saliva samples have been studied by this same procedure to ascertain the effect of the solubility factor of the saliva for calcium and phosphorus on susceptibility to dental caries and whether this factor may be a controlling one in the environment of the tooth which determined the level of immunity. The clinical and experimental results of this study have revealed that, in immunity to dental caries, the inorganic phosphorus and calcium moved from saliva to bone chips and that, in active dental caries, the inorganic phosphorus and sometimes the calcium (either or both) moves from the bone chips to the saliva instead of from the saliva to the bone chips. The discovery of this physicochemical condition immediately threw light on many of the clinical observations; for example, the progressive hardening of the surface of crowns of teeth for several months after eruption, and also on observations regarding the change in den-

sity of tooth structure under such stresses as pregnancy, wasting disease and rapid growth of childhood. These conditions are also favorable for the development of caries. The findings are very important, if they are found to prevail.

As a means for critically checking the application of these new principles, several groups of patients have been investigated. One, a large group, has consisted of clinical patients who had been studied carefully by roentgenographic examination, clinical examination (by instrument) and by chemical analysis of the saliva. Another group consisted of forty children from the Broadway Mission selected because of very rampant dental caries, their nutrition being very bad owing to the stress of the industrial depression. Many of these children had caries extending to the pulp chambers, though the pulps were still vital. The pulps were protected in most of these teeth with temporary fillings, and careful clinical inspection was made together with roentgenographic studies. These children received as treatment one well balanced meal a day especially reinforced in mineral carrying foods and high vitamin butter. This meal was commenced by each child in line taking a half a glass of tomato or citrus fruit juice and a teaspoonful of half and half of a high vitamin cod liver oil and high vitamin butter concentrate mixture provided by me. They then proceeded to the table and had a large bowl of vegetable and meat soup containing a varied assortment of vegetables and chopped meat prepared as stew. Many of the children took a second liberal helping of soup. For the next course, they had sliced canned peaches and delicious rolls made of freshly ground whole wheat to which had been added, on some occasions, an additional small quantity of fresh wheat germ. (This has been discontinued.) The whole wheat was ground in a motor driven coffee mill and prepared freshly from day to day. These rolls were spread liberally with an extra high vitamin butter, which had been produced by cows while pasturing on green wheat. Each child had also one or two glasses of milk. This meal was varied from day to day, but so designed as to maintain these high levels of fat-soluble activators, minerals and grain embryo. The children received six meals a week. The source of vitamin C was varied. Carotene was reinforced by adding to the vegetable soup finely ground small carrots of a deep yellow. These and the wheat germ were added to the vegetable soup after it was removed from the stove but still hot, to avoid the high temperature and long cooking, which would injure the activator content. This meal provided 1.48 gm. of calcium and 1.28 gm. of phosphorus in a single helping.

A third study group consisted of patients selected by groups of dentists in about a dozen cities each providing a patient with a history of marked susceptibility to dental caries. Control of the condition was undertaken by sending to these selected patients some high vitamin product and instructions relative to the reinforcement of the daily nutrition with high mineral carrying foods. In all of these groups, the average movement of inorganic phosphorus was in the wrong direction before treatment and in the right direction when the second sample of saliva was procured one or two months later. Additional roentgenograms were made from time to time, and in only one case was dental caries reported to be in progress.

In many cases, in all three groups, there was marked roentgenographic evidence of improvement in the density

of the teeth, in reduction and size of the pulp chamber and in the building in of protective walls underneath the caries. Illustrations of these structural changes have been presented.[1]

These and other data have suggested the urgent need that these new principles be checked in each group of individuals living in natural conditions which provide immunity and other groups living in conditions providing susceptibility, if such could be found living under very exacting nutritional conditions and where the diets could be studied and analyzed. This cannot be done with diets of those earlier civilizations and primitive people whose skeletons reveal complete absence of dental caries. This has constituted an important part of the new investigations a preliminary report of which is included in this paper and a brief résumé of which has been made of studies conducted by me in Switzerland in 1931.

Since human nutriment in various parts of the world has contained a few principal food factors, there has been need for more detailed information regarding the variations in the mineral content of the same foods produced under different conditions and in different localities. This has been done both by chemical analysis of the foods and by studying the effect of these variations on experimental animals when detailed analysis could be made of the chemical content of tissues as well as minor and gross structural changes. This will throw light on the dietary studies of the experimental groups and will be presented first.

It will assist in visualizing the problem if we will first state briefly the principal factors that have seemed to be established. Arthur D. Black,[2] in summing up the work of W. D. Miller, J. Leon Williams and G. V. Black, says:

> In brief, the facts are: 1. The structure of the tooth, except in gross defects such as pits and fissures, has little or no relation to the occurrence of decay. . . . 2. Caries always begins on the surface of the tooth or in a defective pit or fissure in the enamel and gradually progresses inward. 3. Caries practically always begins in certain protected spots and spreads on the surface within definite limits which are exactly definable as the area of liability for each surface. 4. Caries practically never begins elsewhere on the surface of a tooth, and the dentin is never involved until the enamel has been penetrated. 5. The dissolution of the structure of the tooth is caused by an acid formed by micro-organisms which are protected by a gelantinous plaque in such a way that the acid is confined to the particular spot and not allowed to become dissolved in the saliva. 6. The acidity of the saliva has nothing to do with the occurrence of decay.

Robert Kesel[3] summarizes past and present theories as follows:

> From a review of the past experiments and observations, what do we actually know about this disease, dental caries? We possess these facts:
>
> 1. Primitive people both ancient and modern, living in their native environment, developed little decay.
>
> 2. With the advancement of civilization with the refinement of food and living conditions, dental caries has markedly increased.
>
> 3. Caries is, as a rule, most active during childhood and adolescence; that is, during the growth period.
>
> 4. Metabolic disturbances produced by such conditions as pregnancy and diabetes seem to increase susceptibility to caries.
>
> 5. The disease has never been produced outside the oral environment.
>
> 6. It stops with the extraction of the tooth and ceases with the death of the individual. Teeth and skeletons are not subject to proteolytic decomposition as is the rest of the body.
>
> 7. The crowns of extracted teeth mounted

2. Black, A. D.: Future Practice of Operative Dentistry, J.A.D.A., 19:1213-1221 (July) 1932.

3. Kesel, Robert: What Do We Know About Dental Caries? (A Critical Review of Recent Investigations), J.A.D.A., 19:903-917 (June) 1932.

on artificial dentures and worn in susceptible mouths sometimes develop caries.

8. Caries is confined largely to so-called areas of susceptibility. These are more or less protected areas, where there is opportunity for stagnation and the collection of foreign material. The more exposed areas, such as prominent angles and cusps, are immune.

9. Caries does not begin or extend under healthy gum tissues.

10. Aciduric and acidogenic bacteria can be found in the active carious cavity.

11. These organisms can be cultured from the saliva of the susceptible individuals, yet not all susceptible areas in the mouth become carious, although exposed to the same bacteria. Of two approximating teeth, one may be destroyed by the carious process and the other not even be affected.

12. The caries lesion, although developing in a susceptible area, may become arrested and remain so indefinitely.

It is of interest that both of these reviewers refer to the available information as they have summarized it as "facts."

Many investigators have laid much stress on the hardness or softness of foods, assuming that the former is necessary, yet large immune groups have existed whose diets have consisted of very soft and sticky foods. Hoppert, Webber and Canniff[4] have recently presented data which they interpret to indicate that the coarseness or fineness of the cracked corn in an otherwise similar diet has made the difference between a very high and a very low incidence of dental caries in rats. Klein and McCollum,[5] in discussing previously presented data, including that of Hoppert, Webber and Canniff, have interpreted the controlling factors as being involved in the levels and ratio of calcium and phosphorus as concerned in the development of dental caries in rats. They say:

1. Estimations of the phosphorus content of diets reported to produce caries in rats indicate that rations containing 0.4802 gm. of phosphorus per hundred grams of diet, or less than 0.4802 gm. of phosphorus per hundred grams of diet, tend to induce dental caries in rats if Ca intake is 0.3424 per cent.

2. Estimations of the phosphorus content of diets reported to produce rats immune to dental caries indicate that such diets contain 0.5282 or more grams of phosphorus per hundred grams of diet and 0.4012 or less grams of calcium per hundred grams of diet.

During the past decade, I have been making a study of the effect of various modifications of nutrition on the incidence of dental caries. The results have been presented in many reports.[6] These have been reviewed in an article entitled "Control of Dental Caries and Some Associated Degenerative Processes Through Reinforcement of the Diet with Special Activators."[7] Data are there presented indicating that dental caries has been controlled by these procedures to about 90 per cent by reinforcing the nutrition with fat-soluble activators in natural form and high mineral foods in natural form. Many cases were reported in which, before treatment, there were as many as two or three dozen cavities, and after or during

4. Hoppert, C. A.; Webber, P. A., and Canniff, T. L.: Production of Dental Caries in Rats Fed Adequate Diet, J. D. Res. 12: 161-173 (Feb.) 1932.

5. Klein, Henry and McCollum, E. V.: Science, 74:662 (Dec. 25) 1931.

6. Price, W. A.: New Light on Some Relationships Between Soil Mineral Deficiencies, Low Vitamin Foods and Some Degenerative Diseases Including Dental Caries, with Practical Progress in Their Control, Bull. Indiana D. Soc., September, 1932; Footnote 1; Are Activators Revealing Nature of Life in Health and Disease Including Dental Disease? Ohio D. J., August-November 1932; New Light on Cause and Prevention of Dental Caries and Other Degenerative Diseases, Proc. Eighth Internat. D. Cong., Paris, 1931.

7. Price, W. A.: J.A.D.A., 19:1339 (Aug.) 1932.

treatment with reinforced diet, they had either no cavities or one or two a year.

These studies have presented much evidence that phosphorus is a mineral that is frequently needed in larger amounts and, further, that its shortage in easily available natural foods is in part associated with and in part apparently controlled by the exhaustion of phosphorus in the soil. This latter phase has been discussed in another communication.[8]

An important phase of my investigations has been concerned with the variation in levels of natural food vitamins with the seasons and as influenced by geographic location, climate, altitude, etc. Seasonal cycles seem to have an important relationship to the seasonal incidence of many diseases, including dental caries. They further indicate that the vitamin levels of the butter-fat of milk as produced in various countries in the world and which show a seasonal cycle are directly related to the type of food being eaten by the cows, goats and other dairy animals. Among the foods found to be most favorable for the production of high vitamin levels have been rapidly growing young green wheat and rye.

The investigations herewith reported will accordingly cover (1) comparative levels of minerals in different cereal foods and the same cereal foods as produced in different places; (2) results from feeding fifteen different diets to fifteen groups of rats; (3) structural changes produced in human teeth by reinforced diet and (4) results of field studies of districts providing immunity and districts providing susceptibility to dental caries including chemical analyses of saliva.[8]

EXPERIMENTAL DATA

1. *Comparative Levels of Minerals in Different Cereal Foods and the Same Cereal Foods When Produced in Different Places.*—Since different sections of the earth provide conditions which are especially suitable for certain cereal plants and unsuitable for others, the available food of different districts is essentially different, the people living in those communities being limited to that particular cereal. It is important that we know the mineral content of the cereals used in the animal experiments to be reviewed presently.

Data presented in Table 1 represent the averages of a number of determinations for each of the five cereals, corn, rye, barley, wheat and oats. Only the calcium, phosphorus, iron and copper content is shown in this table. It will be seen that the calcium level shown as the percentage of calcium oxide in grain is highest in oats, but the percentage in ash is highest in corn. The phosphorus as pentoxide percentage in grain is the highest in barley and more than twice as high as in corn. Indeed, the phosphorus of the corn is only about half as high on an average as in each of the other four cereals. This table may be very misleading unless we consider carefully the variations that can take place in the different samples of the same cereal which have grown in different places at the same time, the controlling factors being local.

A study for example of nine samples of wheat obtained on the market showed a variation in phosphorus content of over fourfold. So great a variation as this is not common. Through the assistance of the department of agriculture in Canada, I have received samples of the same marquis wheat grown in about twenty places in Canada. Data on seventeen of these are reported in Figure 1. The places are shown below each mention of calcium, phosphorus, potassium, magnesium, iron

8. Footnote 6, first reference.

and protein. This is helpful in several ways, showing at a glance the relative amount of these minerals and the protein in wheat and, by the difference in the height of the columns, the variations that were found by growing this sample of wheat in seventeen places. At once we see that wheat carries a high percentage of phosphorus in comparison with the other minerals, particularly as compared with calcium. The animal body's need for iron is imperative. The greatest variation of the minerals disclosed in the chart is found in the iron. The difference in the iron content in the last two samples of wheat is a little over fifteenfold. The significance of this will be discussed later. Further data will be presented on the mineral content of different diets containing cereals under the next heading.

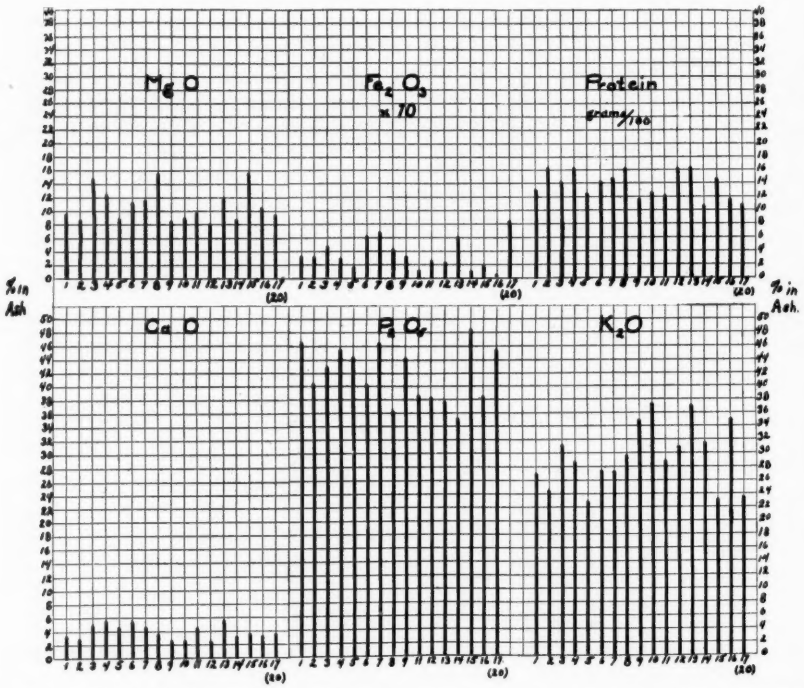

Fig. 1.—Variations in mineral and protein content of the same Marquis wheat grown in seventeen different places in Canada, in 1929. The seventeen experiment stations of the department of agriculture were: *1,* Brandon, Manitoba; *2,* Swift Current, Saskatchewan; *3,* A 12, Plot 13, Swift Current, Saskatchewan; *4,* A 12, Plot 14, Swift Current, Saskatchewan; *5,* Fredericton, New Brunswick; *6,* Lacombe, Alberta; *7,* Fort Vermillion, Alberta; *8,* Indian Head, Saskatchewan; *9,* Ste.-Anne-De-La-Pocatiere, Quebec; *10,* Nappan, Nova Scotia; *11,* Cap Rouge, Quebec; *12,* Agassiz, British Columbia; *13,* Scott, Saskatchewan; *14,* Charlottetown, Prince Edward Island; *15,* Rosthern, Saskatchewan; *16,* Invermere, British Columbia; *17,* (20), Kapuskasing, Ontario.

2. Results from Feeding Fifteen Different Diets to Fifteen Groups of

Rats.—These diets have been planned on a basis that would make possible the comparison of the same cereals obtained from two different sources; namely, two kinds of corn and two kinds of wheat and a modification of diets built on these cereals with activators and with different parts of the grain. It has been deemed important to make this study in order that detailed chemical analyses might be made of blood and other body tissues, as well as a study of the teeth for dental caries. factors used were on the same basis of separation with a twenty mesh screen as suggested by Hoppert, Webber and Canniff. (*c*) The influence of activators of different sources. (*d*) The use of inorganic tricalcium phosphate. (*e*) Comparisons of the different wheat products as provided in the whole wheat, the flour, the bran and middlings mixture. For these studies, fifteen groups of rats were provided consisting of from four to seven rats each. These were obtained from four different

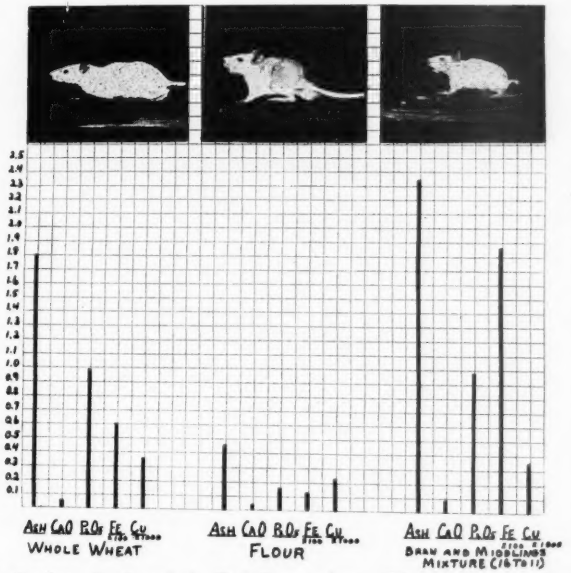

Fig. 2.—Distribution of minerals in Red Cross wheat. The rats were fed on these three different wheat products. Four gave marked evidence of lack of minerals.

In planning this series of animal investigations, the following factors have been deemed important: (*a*) A comparison of different sources of the same food. For this, a feed corn of cheaper market value and a seed corn of a higher market value have been used. The wheats used were grown in two different localities. (*b*) A consideration of the relation of the coarseness and fineness. The corn places in order to get as large a number of rats of approximately the same age. Each of these sources was represented in each of the first ten groups in one series and in each of five groups in a second series. The rats in these two groups differed in age, the first group averaging 8 weeks of age and the second group averaging 4 weeks of age at the beginning of the test. The feeding program for

each of the fifteen groups was as follows. The variable factor was added as 69 parts to a basic diet of wheat gluten, 20 parts, whole milk powder, 5 parts, crisco, 5 parts, and sodium chloride, 1 part, as used by Hoppert, Webber and Canniff.

Group 1 received cracked feed corn, which included both the coarse and fine, with the following distribution of particles, 45 per cent remaining on a 20-mesh screen as used by Hoppert, Webber and Canniff. Group 2 received the fine corn, which went through the 20-mesh screen; Group 3, the coarse particles, which remained on the 20-mesh screen; Group 4, freshly cracked whole wheat with the mill at the same set; Group 5, the same as Group 1, namely, both coarse and fine corn meal plus a high vitamin butter *ad libitum;* Group 6, the same as Group 1, namely, both the coarse and fine corn meal plus 2 per cent of a mixture of a high vitamin butter and a high vitamin cod liver oil; Group 7, coarse and fine corn meal as in Group 1, plus 2 per cent of a mixture of high vitamin butter and a high vitamin cod liver oil and 10 per cent of wheat germ; Group 8, coarse and fine feed corn meal as in Group 1, plus 10 per cent wheat germ; Group 9, whole wheat plus 10 per cent wheat germ; Group 10, coarse and fine corn meal plus 10 per cent of dicalcium phosphate. The rats in Groups 11 to 15 inclusive were as stated younger than those in Groups 1 to 10 inclusive. Number 11 received the coarse screenings of a better grade of corn, sold as seed corn, otherwise corresponding with Group 3 plus the basic diet. Group 12 had fine screenings from seed corn as in Group 2. Group 13 received cracked Red Cross whole wheat. Group 14 received Red Cross flour and Group 15, received a Red Cross bran and mid-

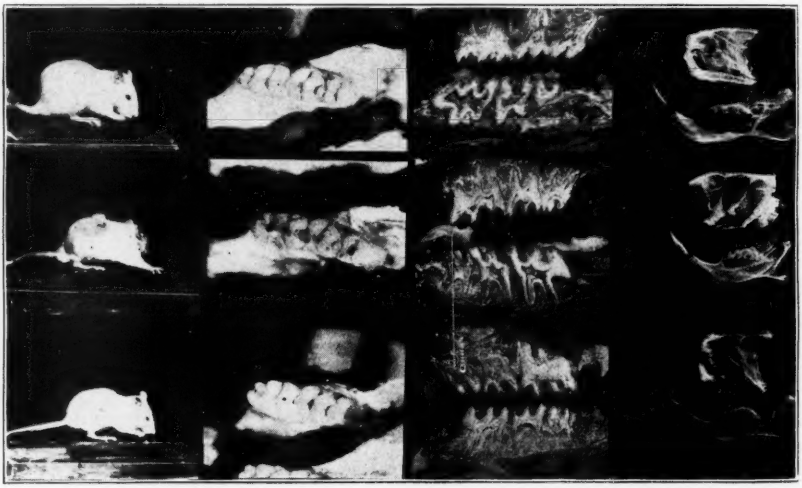

Fig. 3.—Red Cross wheat studies. Above: Group 13, fed stock diet plus whole wheat (all of minerals); center: Group 14, fed stock diet plus wheat flour (one-fifth of minerals); below: Group 15, fed stock diet plus bran and middlings (four-fifths of minerals). The teeth of the rats on the wheat flour supplement showed active caries.

dlings mixture as separated from Red Cross flour; all in addition to the basic diet. One hundred pounds of wheat was said to produce 73 pounds of flour, 16 pounds of bran and 11 pounds of middlings. (The miller's report.)

The last three groups have been particularly interesting because of the need for the greatest possible efficiency in the wheat ration; 89 per cent for Group 14, receiving the flour of the wheat, and 112 per cent for Group 15, receiving the middlings and bran mixture. There was a great difference in the appearance of the animals, particularly in the fur coat which is illustrated in Figures 3 and 4. As is readily seen, the fur of these rats in the flour group came out in patches

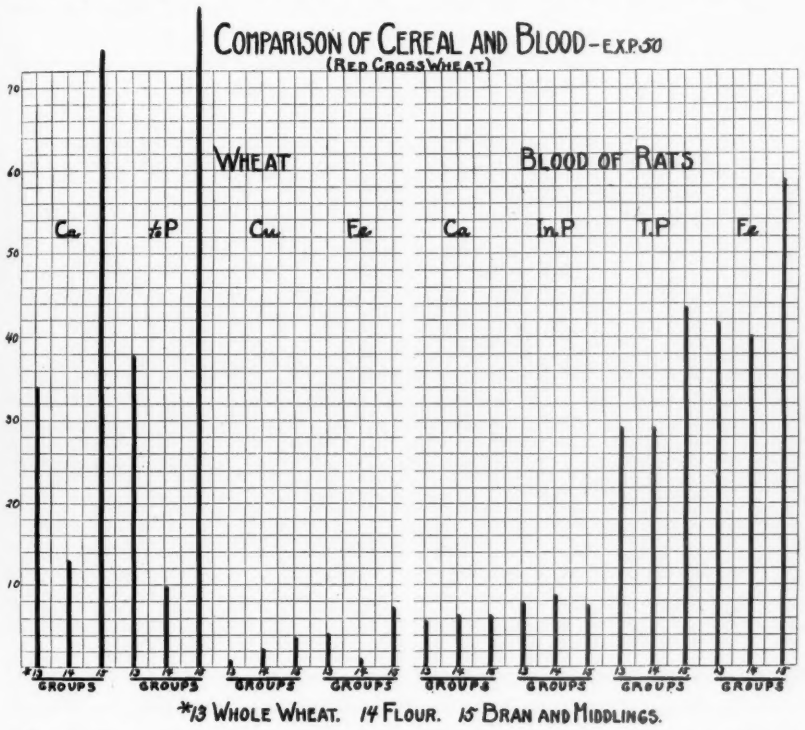

Fig. 4.—Deficiency of food minerals again demonstrated in chemical content of blood of rats.

planning of dietaries during this strenuous industrial depression. These three groups will be reviewed first.

The percentage gain in weight of these three groups after seven weeks of the feeding program was 154 per cent for Group 13, receiving the cracked whole and looked very bad and they lacked the "pep" and energy of those in the other two groups. When a finger was snapped or any other sudden noise was made, rats on the flour jumped perceptibly as an involuntary reaction. This did not occur with either of the other two groups.

The mineral content of the Red Cross wheat, flour and bran and middlings, which were furnished to me by a miller who ground the wheat for the Red Cross, is presented in Table 2.

It is of interest to note that four fifths of the ash went with the middlings and bran. There was 2.26 times as much calcium and 5.26 times as much phosphorus provided in the whole wheat of a given weight as in the flour of that weight. The bran and middlings mixture provided 4.53 times as much calcium for a given weight as flour and 6.1 times as much phosphorus. These data are shown graphically in Figure 2, with the photographs of a typical rat from each of the three groups; namely, those receiving whole wheat, those receiving flour and those receiving bran and middlings mixture as a supplement to the basic ration. This chart should be read vertically. It is important that these figures and their significance be kept in mind, since it is not easily possible under ordinary conditions for most people to eat enough additional white flour products as a means of obtaining their required minerals to make up what has been lost by the removal of the bran and middlings. That this is true for the experimental animals used is emphasized by the fact that those receiving foods low in minerals, the flour mixture, ate less food instead of more. The physical differences which are so strikingly brought out in the photographs in Figures 2 and 3 were associated with a very marked difference in the structure of the various organs and tissues, including the teeth and their supporting structures. A detailed study of these seems to throw important light on the problems of the etiology and control of dental caries. The mineral content of diets 13, 14, and 15 is shown in Figure 2, for comparison. Note the wide variation.

Table 1.—Comparison of Mineral Content of Different Kinds of Grain

		Calcium Oxide in		Phosphorous Pentoxide in		Ferric Oxide in		Copper in	
	Ash Per Cent	Ash Per Cent	Grain Per Cent	Ash Per Cent	Grain Per Cent	Ash Per Cent	Grain Per Cent	Ash Per Cent	Grain Per Cent
Corn	1.26	4.21	0.053	25.3	0.319	0.153	0.00259	0.0041	0.000069
Rye	1.81	2.49	0.045	34.7	0.600	0.568	0.0089	0.00176	0.000278
Barley	2.16	2.08	0.046	32.5	0.724	0.879	0.0188	0.0027	0.000057
Wheat	1.70	2.82	0.048	38.0	0.655	0.332	0.006	0.0198	0.00036
Oats	2.98	3.03	0.089	20.7	0.608	0.000	0.000	0.0000	0.00000

A series of studies of the teeth and their supporting structures are shown in Figure 3. In this chart, the data for each group are arranged horizontally. The first column shows another typical view of the rats of the three different groups. The second column shows photographs of the occlusal surface of the teeth. In these, it will be seen that the crowns are well formed in all three series. The second group, those on the white flour, show cavities extending into the teeth on the

occlusal surfaces. The sulci of rats' teeth are normally quite deep. The third column shows photographs of the ground sections of the teeth of each of the three groups. The second group shows marked destruction by caries of the crowns, approaching the pulp in some of the cavities. The first and third group are practically normal. The fourth column shows roentgenograms of the teeth which were prepared by first soaking the specimens in silver nitrate, a 10 per cent solution, for a few hours, then exposing them to light. In this condition, decalcified tooth structure which would normally be quite transparent to the roentgen-rays, becomes more opaque owing to the presence of metallic silver.

In Figures 2 and 3, I have shown the relationship between the physical conditions of the rats in the different groups, this depending on whether they received whole wheat, flour or the bran and middlings mixture. The difference in content of some of the chemicals in each of these three forms of wheat cereal and a comparison of the structure of the teeth of the rats fed on these three diets has been shown. These studies have also included a comparison of the levels of several of the minerals in the blood of rats on different diets with the difference in the levels of the minerals in the different diets. This is illustrated in Figure 4.

It will be noted that the calcium and inorganic phosphorus of the blood is nearly the same in all three groups. There is a marked difference in the total phosphorus of the blood, it being much higher in Group 15 than in Group 13 or 14. The total phosphorus in the wheat is also much higher in Group 15, which is the bran and middlings mixture, than in the whole wheat group, and very much higher than in the flour group. The most striking difference is in the iron content of the three foods and the three specimens of blood, it being much lower in the flour than in either the whole wheat or bran and middlings mixture and much higher in the blood of the rats in this group and lower in the wheat flour group than in the whole wheat group. The significance of these data will be much more apparent when we come to the discussion of this summer's studies of human beings showing immunity or non-immunity to dental caries.

TABLE 2.—MINERAL CONTENT OF RED CROSS WHEAT, FLOUR AND BRAN AND MIDDLINGS

	Ash	Calcium	Calcium as Calcium Oxide	Phosphorus	Phosphorus as Phosphorus Pentoxide	Iron	Copper
Whole wheat	1.8	0.034	0.048	0.38%	0.974	0.006	0.00036%
Flour	0.47	0.015	0.021	0.07%	0.159	0.0013	0.00022%
Bran and Middlings Mixture	2.34	0.068	0.095	0.43%	0.981	0.0187	0.00035%

The foregoing data have related to the last three groups of rats in the series of fifteen diets previously outlined. Several chemical factors in addition to these here presented have been determined on the blood of the rats of these fifteen series. This has been done by pooling the blood of the different rats in the groups. The levels of the various chemicals are shown in parallel columns for comparison in Figures 5, 6, 7 and 8. The remarkable influence of diet on some of the

blood chemical factors is immediately made apparent by a comparison of the different heights of the columns.

In Figure 5 are shown the levels of each calcium, the percentage of change in calcium when powdered bone chips were added to the blood serum and percentage of calcium in the cells. Diets 1, 2 and 3, representing the feed corn, fine feed corn and coarse feed corn, respectively, are of special interest and should be noted in by Hoppert, Webber and Canniff.[4] The blood serum calcium was slightly higher in the group of rats receiving fine feed corn. The percentage change of calcium plus bone was lowest in the group receiving the coarse feed corn, and the percentage of calcium in the blood cells highest in the group receiving the whole corn (both fine and coarse), and lowest in the group receiving the coarse feed corn. These changes were not nearly so

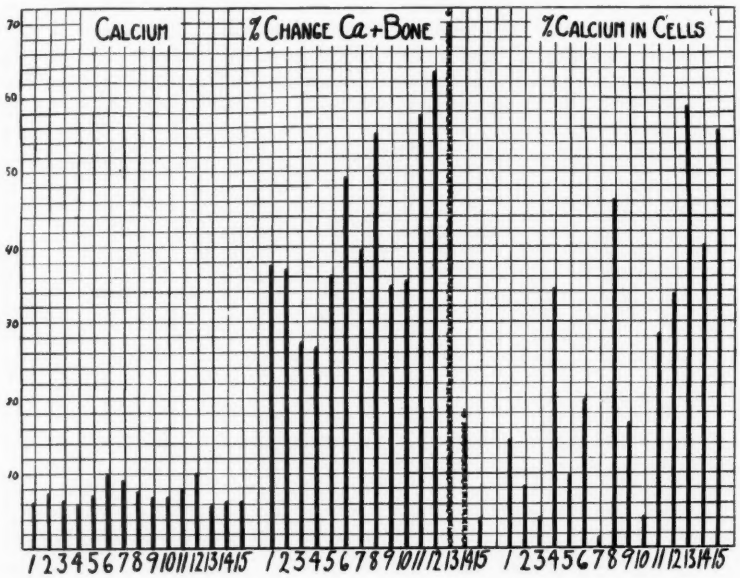

Fig. 5.—Blood chemical changes of fifteen diets. Comparisons in three blood factors: calcium, percentage of change of calcium of serum plus bone and percentage of calcium in cells of rats fed on fifteen different diets. The crosses indicate a reverse direction of movement of calcium in the center group. The diets were: *1,* fine and coarse feed corn; *2,* fine feed corn; *3,* coarse feed corn; *4,* fine and coarse whole wheat; *5,* No. 1 plus high vitamin butter; *6,* No. 1 plus 1 per cent of cod liver oil and of high vitamin butter; *7,* No. 6 plus 10 per cent wheat germ; *8,* No. 1 plus 10 per cent wheat germ; *9,* No. 4 plus 10 per cent wheat germ; *10,* No. 1 plus dicalcium phosphate; *11,* coarse seed corn; *12,* fine seed corn; *13,* Red Cross fine and coarse whole wheat; *14,* Red Cross flour; *15,* Red Cross bran and middlings.

these various data because of the suggestion that the coarseness of the corn was the cause of decay in the series reported great as the changes produced by some other factors, as, for example, the effect of adding dicalcium phosphate to the

diets in Group 10 and an excess of wheat germ in Group 7.

Figure 6 shows, for comparison, the inorganic phosphorus, the per cent change in inorganic phosphorus when powdered bone chips were added to the blood serum and the percentage of phosphorus in the cells. In observing these different levels, it is important to observe that two different kinds of corn, one a low grade commercial feed corn (used in diets 1, 2 and 3), the second a high grade commercial seed corn (used in diets 11 and 12); also wheat from two different sources, one used in diet 4, and the other in diet 13, produced quite different effects when fed; which suggests that it would not be justifiable to apply these readings for interpreting data presented by other workers using another sample of corn or wheat. It should be noted particularly in Figure 6 that the phosphorus of the blood cells was exceedingly low in rats from Group 14 receiving the white flour and high in Groups 13 and 15, receiving the whole wheat and the bran and middlings mixture. The phosphorus of the cells of the groups receiving the fine sift-

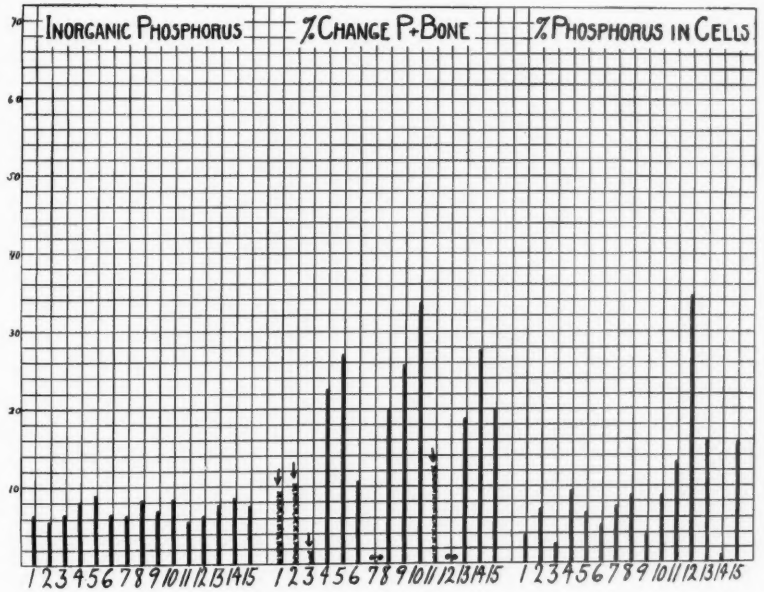

Fig. 6.—Blood chemical changes on fifteen diets. Comparisons in three blood factors: inorganic phosphorus, percentage of change of phosphorus of serum plus bone and percentage of phosphorus in cells of rats fed on fifteen different diets. Crosses indicate a reverse direction of movement of phosphorus in the center group. The diets were: *1*, fine and coarse whole feed corn; *2*, fine feed corn; *3*, coarse feed corn; *4*, fine and coarse whole wheat; *5*, No. 1 plus high vitamin butter; *7*, No. 6 plus 10 per cent wheat germ; *8*, No. 1 plus 10 per cent wheat germ; *9*, No. 4 plus 10 per cent wheat germ; *10*, No. 1 plus dicalcium phosphate; *11*, coarse seed corn; *12*, fine seed corn; *13*, Red Cross fine and coarse whole wheat; *14*, Red Cross flour; *15*, Red Cross bran and middlings.

ings of feed corn has the highest level. The percentage change in inorganic phosphorus when bone chips were added to the blood serum will be discussed later.

In Figure 7 will be seen a comparison of the levels of potassium, iron and total phosphorus for the different groups. It should be noted that the comparison of the level of potassium with that of iron and of phosphorus in this chart requires the explanation that the potassium colcance of a comparison of the potassium in the blood of each of the fifteen groups of rats which shows a difference of over 100 per cent between Group 4 and Group 12. There is also a marked difference in the level of the iron and total phosphorus in the blood of the different groups.

In Figure 8 will be seen a comparison of the cell volume, the hemoglobin and the gain in weight of the different groups. As already stated, the rats in the last five

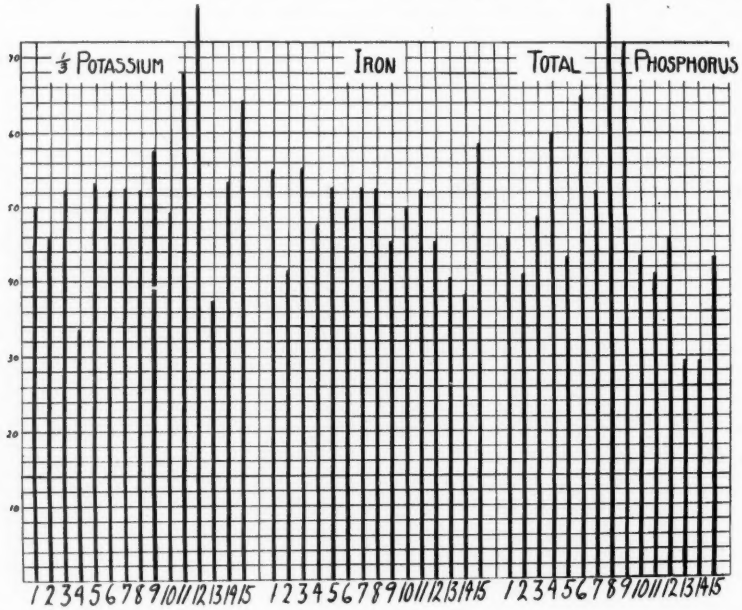

Fig. 7.—Blood chemical changes on fifteen diets. Comparisons in three blood factors: potassium, iron and total phosphorus of rats fed on fifteen different diets: *1,* fine and coarse feed corn; *2,* fine feed corn; *3,* coarse feed corn; *4,* fine and coarse whole wheat; *5,* No. 1 plus high vitamin butter; *6,* No. 1 plus 1 per cent each cod liver oil and high vitamin butter; *7,* No. 6 plus 10 per cent wheat germ; *8,* No. 1 plus 10 per cent wheat germ; *9,* No. 4 plus 10 per cent wheat germ; *10,* No. 1 plus dicalcium phosphate; *11,* coarse seed corn; *12,* fine seed corn; *13,* Red Cross fine and coarse whole wheat; *14,* Red Cross flour; *15,* Red Cross bran and middlings.

umns are one-third their proper height as shown because of the large amount of potassium in comparison with other minerals. This does not change the signifigroups, namely, 11, 12, 13, 14 and 15, were younger than those in the first ten groups and show a more rapid growth than those in the first ten groups. The

rate of growth progressively lessens with increasing age. The greatest gain in weight occurred in the rats receiving whole wheat in the second series and the lowest gain in weight in the first series in the rats receiving fine feed corn. It is important to observe that the two different grades of wheat used in Groups 4 and 13 produced very different growth characteristics. The hemoglobin of the blood shows a marked variation in different iron of the bloods was in direct proportion to the iron in the food of the different groups.

An important phase of this study of the effect of fifteen different diets on the groups of rats has related to the incidence of dental caries. It is difficult to compare the data provided by different workers, chiefly because of different procedures of study. The results that are shown in Figure 9 represent cavities per rat.

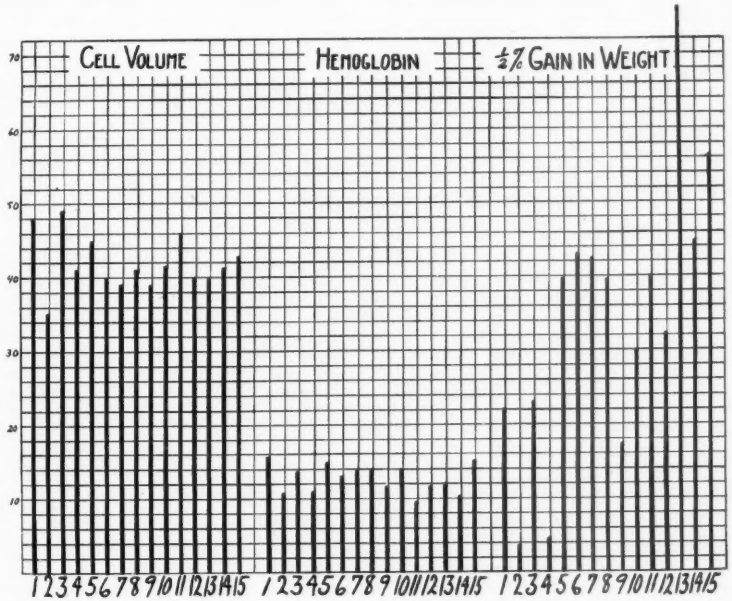

Fig. 8.—Blood chemical changes on fifteen diets. Comparisons in two blood factors, cell volume and hemoglobin, and gain in weight of rats fed on fifteen different diets: *1,* fine and coarse feed corn; *2,* fine feed corn; *3,* coarse feed corn; *4,* fine and coarse whole wheat; *5,* No. 1 plus high vitamin butter; *6,* No. 1 plus 1 per cent of each cod liver oil and high vitamin butter; *7,* No. 6 plus 10 per cent wheat germ; *8,* No. 1 plus 10 per cent wheat germ; *9,* No. 4 plus 10 per cent wheat germ; *10,* No. 1 plus dicalcium phosphate; *11,* coarse seed corn; *12,* fine feed corn; *13,* Red Cross fine and coarse whole wheat; *14,* Red Cross flour; *15,* Red Cross bran and middlings.

groups and will be seen to vary in almost exact relation to the iron of the feed as shown in Figure 2. I have previously shown that in Groups 13, 14, and 15, the

When staining methods and histologic sections are used, the microscope will reveal structural changes in very minute proportions, which cannot be observed or

estimated by less exacting procedures. I am not reporting the data obtained with the assistance of the high magnification microscope. Cavities that were sufficiently large to be seen with the naked eye or with the low power of the microscope are indicated by the column. The number of cavities per rat which had entered into the pulp chambers is indicated by a cross line with a letter P. It will at once be seen that there is a marked difference in the number of cavities produced in the rats on these different diets, and it is of particular interest that the data produced by these studies have not developed the same proportion of cavities whether in the groups eating coarse corn or in those eating fine corn, as reported by Hoppert, Webber and Canniff. The significance of these data will be discussed presently. It is important to note that Groups 4, 5, 6, 9 and 15 had no caries and Group 13 almost complete immunity. The rôle of the activators in conjunction with the mineral carrying foods is strikingly indicated in Groups 5 and 6.

3. *Structural Changes Produced in Human Teeth by Reinforcement of the Diet.*—If dental caries is fundamentally controlled in its major aspect by physicochemical factors of the saliva, which, in turn, are controlled by nutrition, it should be possible to produce changes in caries cavities, even after they have been established, by nutritional means. In Figure 10 will be seen two teeth, both deciduous molars. The one to the left was removed from a child with active dental caries, without treatment. After extraction, the tooth was immersed in a 10 per cent solu-

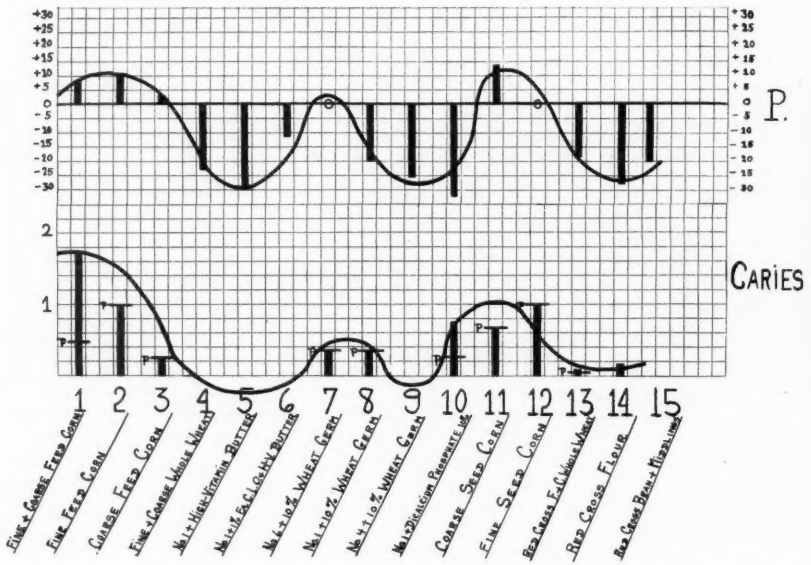

Fig. 9.—Dental caries in rats on fifteen diets (below); solubility factor of blood for phosphorus (above). The inorganic phosphorus of the blood serum of normal animals moves to bone chips. The tendency to follow the incidence of caries is shown below for the different diets.

tion of silver nitrate for several hours, then placed in sunlight. The ground surface reveals that the soft caries portion has been completely saturated with silver nitrate. Other parts of the tooth have not been penetrated, except a small proximal area of caries on the opposite contact. The tooth to the right shows a similar tooth which was left in the mouth during twelve weeks of treatment, being a tooth from one of the children of the Broadway Mission group. This tooth was treated similarly to the one at the left, and it is important to note that the silver nitrate had not penetrated the carious structure much more than the

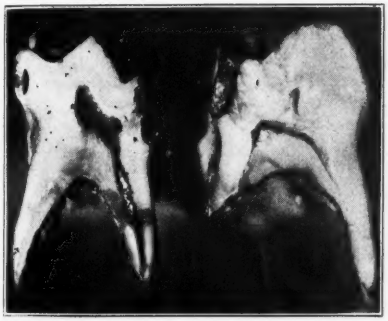

Fig. 10.—Left: Tooth showing silver nitrate saturated to depth of carious dentin; from a patient without treatment. Right: Tooth showing caries controlled. The silver nitrate has not been able to penetrate much if any farther than into the enamel surface of the tooth.

enamel surfaces of the tooth. To the point of a sharp instrument such as an explorer, this surface was glassy hard. It is routine practice to leave many such teeth unfilled for critical examination. The result shown in this tooth is typical of large numbers so examined.

An opportunity for a more severe test is provided in teeth where the decalcification of large occlusal and proximal cavities has reached the pulp, and where it would be expected that toxic material, if not actual bacterial invasion, had already entered into the pulp tissue. Such a case in which a large area of dentin over the pulp had been decalcified is shown in Figure 11. Without removal of the carious dentin, a temporary cement filling was placed in the cavity to prevent pressure and packing of foods. The patient was one of the Broadway Mission group receiving one reinforced meal a day. The condition at the beginning is shown to the left, and on the right is seen the result after about twelve weeks on the reinforced diet. The coronal aspect of the pulp has now been protected by a deep layer of newly laid down dentin. The exposed pulp is no longer exposed. Of twelve teeth with far advanced dental caries apparently involving the pulp as in the case here presented, two became abscessed and ten showed marked structural change by building in of dentin in the pulp chamber to provide a protecting wall. In some cases the teeth were purposely left without fillings and the caries did not proceed with a breakdown of the pulp tissue.

4. *Results of Field Studies of Districts Showing Immunity and Districts Showing Susceptibility to Dental Caries, Including Chemical Analyses of Saliva.*—An explanation of the phenomena of dental caries, to be adequate, must stand testing by application to areas of immunity and areas of susceptibility. Areas in which the people will generally be found to have lost their immunity are very numerous and very easy to find; not so with areas of immunity.

I have reported efforts made last year to find groups with immunity in the high Alps of Switzerland and particularly one large group with a high level of immunity. Accordingly, much effort

has been expended during this summer of 1932 in search of groups with high immunity living under quite exacting conditions, where the controlling factors would be relatively similar for large groups. Extended correspondence relative to different countries led to the decision in favor of Switzerland and the Inner and Outer Hebrides as important fields for this study. In all, about twenty different districts were studied in most of which, in addition to a detailed and clinical study of the mouth and living habits, a sample of saliva was obtained, cultures were made of the flora with special consideration of the acidophilic group and the bacterial growth of the saliva was controlled by the addition of formalin. One hundred and seventy-one samples of saliva were forwarded or brought by me to my laboratories for chemical analysis and arrangements were made for study of other samples in provided containers. In all about forty items of information were obtained for each patient. In addition, twenty chemical factors were provided in the salivary analysis.

The first place studied this summer was the Loetshental Valley, which has an altitude of from 4,000 to 6,000 feet for different parts of the principally occupied valley. These people have had a forced isolation owing to the high snow-capped mountain ranges surrounding the valley with the only entrance a dangerous one owing to the presence of treacherous avalanches in the narrow gorge. Recently, the opening of the Loetschberg tunnel, the second longest in Europe, made a doorway for these people to the outer world, and also permitting the accompaniments of modern civilization to have access to the valley. Even since last year, I have found evidence of modernization both in dress and in foods available. A modern white flour bakery, built within the year, is rapidly influencing the dietary. Compared with most communities, they still have high immunity to dental caries.

Fig. 11.—Control of dental caries under nutritional treatment with building in of new pulpal wall.

This valley has four principal villages connected by a footpath on which the post mules carry the government mails. The transportation of the valley is largely by human beings. All of the farming and local industrial work is done by human power. Sojourns were made to other districts for carrying forward the clinical examinations and obtaining saliva samples for chemical analysis. Studies were also made

in districts in the lower plains country of Switzerland. Switzerland, an inland country, provided a different environment from that near the sea. I, therefore, also made studies in the Inner and Outer Hebrides, north Scotland, Wales and England.

A group of nine places in which the incidence of dental caries was very low are shown in Figure 12 (below, left). To the right are shown seven places where the incidence of dental caries was lifetime as far as could be determined by either the presence of cavities or fillings or missing teeth in the case of young people or where the history of the patient, if older, could decide one that the tooth had been lost by caries. The second column for each place represents the percentage of people who do not have a history of complete immunity from dental caries for their lifetime, which I will refer to as "total individual susceptibility." The third column represents the

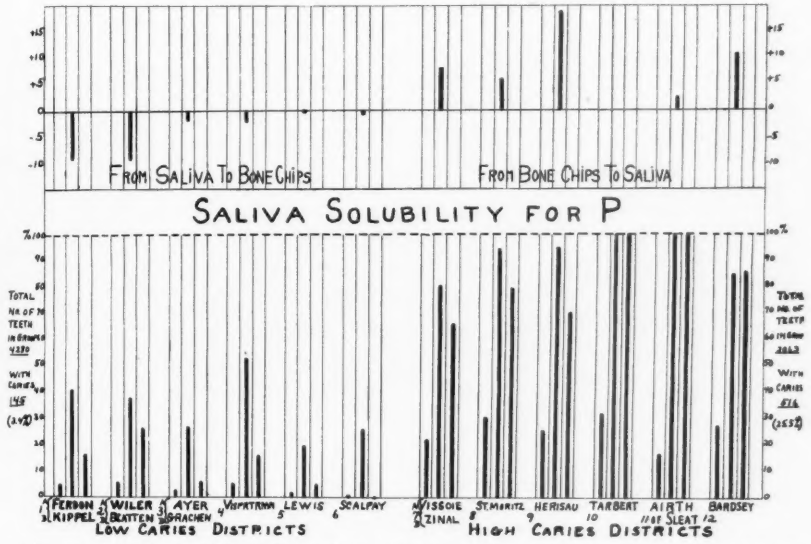

Fig. 12.—Results of field studies both in districts showing immunity and in those showing loss of immunity to dental caries. Above is shown the behavior of the serum inorganic phosphorus, which moves in the normal direction in groups with low caries incidence and in the wrong direction in the groups with a high incidence.

very high. In both groups are included places representing characteristic types of environment with regard to altitude and climatic conditions. For each place, three factors in dental caries are shown graphically by the columns. The first column from the left represents the percentage of teeth that have been involved by dental caries during the individual's entire

percentage of individuals who, at the time of the examination, were free from active dental caries, which I have referred to as "present susceptibility." The first two towns or villages, Ferdon and Kippel, are in the lower part of the Loetschental Valley nearest to the tunnel and railroad station and have an altitude of about 4,000 feet. The second group, Wiler

and Blatten, having an altitude of about 5,000 feet, are farther up the valley nearer to the glaciers. Ayer, with an altitude of 4,800 feet, is in the upper part of the Anniviers Valley, and Grachen, with an altitude of 5,000 feet, is on the mountain side well toward the glacier in the Natervisp Valley. Vispertiminen is high up the mountain side in the Visp Valley. Vissoie is in the Anniviers Valley, 5 miles down the valley from Ayer. Zinal is in the same valley as Vissoie. Zinal is populated in the summer by families from Vissoie. St. Moritz, the famous winter and summer health resort, is in the Oberengadin Valley and is among the streams forming the headwaters of the Danube. Herishau is in the rolling low country of Switzerland near Lake Constance. Lewis is the most northerly island of the Outer Hebrides off the northwest coast of Scotland. Tarbert is the principal port of call for boats in the Isle of Harris. Scalpay is in the Isle of Harris south of the Lewis Island. Airth of Sleat is on the island of Skye. Bardsey is an island off the west coast of Wales. In Figure 12, these are shown in the two general groups marked "low caries districts" and "high caries districts," respectively. In

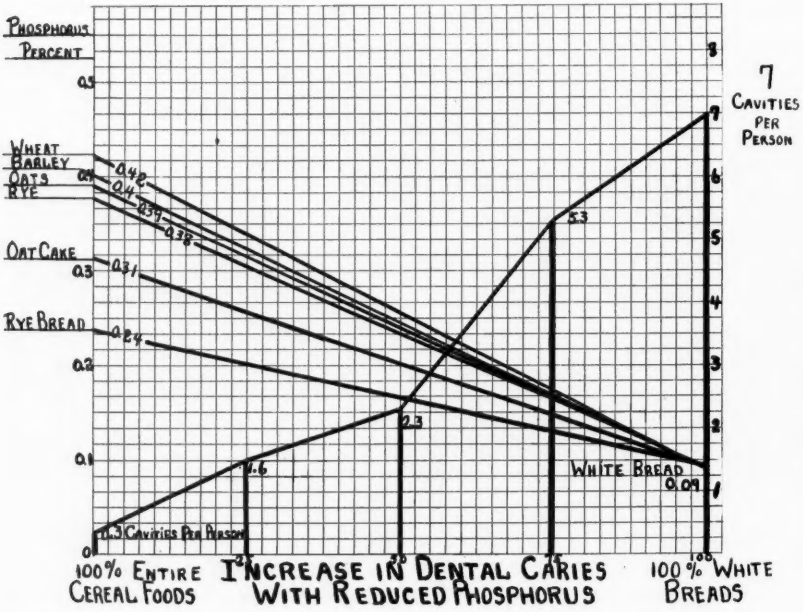

Fig. 13.—Incidence of caries in relation to mineral content of one important food.

the former, 4,080 teeth were examined, of which only 145 had ever been attacked by caries, an incidence of 3.4 per cent on the basis of the number of teeth. In the "high caries districts," 2,063 teeth were examined, of which 516 were attacked by caries, or 25.5 per cent. An effort was made to obtain groups as nearly typical and comparable as possible. For this reason, this study was largely limited to

children under 15 years, in whom the stress period of natural growth would be common.

In Group 1, Ferden and Kippel, in every hundred teeth examined, an average of only 4.8 were found to have been attacked by dental caries (shown in the column to the left). In Group 2, Wiler and Blatten, the average was 5.9, in Group 3, Ayer and Gracken, it was 2.3, and in Group 4, Vispertiminen, it was 5.2. In the Isle of Lewis area, Group 5, Group 9, Herisau, an average of 24.7 per cent; in Group 10, Tarbert, an average of 32.4 per cent; in Group 11, Airth of Sleat, an average of 16.4 per cent, and in Group 12, Bardsey, an average of 27.6 per cent. The general average in this "high caries group" was 25.5 per cent.

The middle column in each group represents "total individual susceptibility"; that is, the percentage of individuals who did not have a perfect history of immunity as indicated by either teeth with

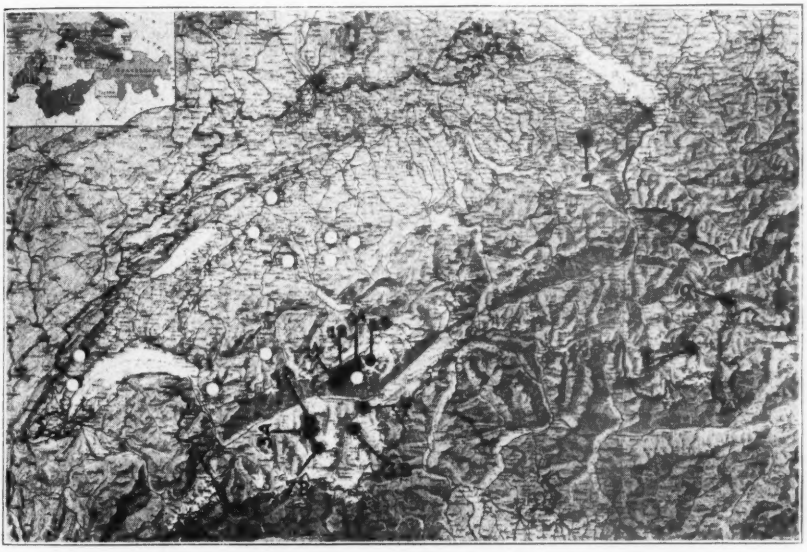

Fig. 14.—Districts in Switzerland where special field studies were made of the incidence of dental caries: *1a,* Ferdon; *1b,* Kippel; *2a,* Wiler; *2b,* Blatten; *3a,* Ayer; *3b,* Grachen; *4,* Visperterminen; *7a,* Vissoie; *7b,* Ziral; *8,* St. Moritz; *9,* Herisau. Butter samples have been received regularly from the places indicated by white dots.

the average was 1.3, and for the Scalpay group number 6, it was 1.0. The general average for the "low caries districts" was 3.4. In the "high caries districts," the percentage of teeth attacked by caries is in Group 7, Vissoie and Zinal, an average of 22.0 per cent; in Group 8, St. Moritz, an average of 29.8 per cent; in caries, filled teeth or missing teeth. For Group 1, this was 40.4 per cent; Group 2, 37.8 per cent; Group 3, 26.6 per cent; Group 4, 35.0 per cent; Group 5, 19.0 per cent; and Group 6, 35.5 per cent; an average for this group of 32.2 per cent. For the "high caries district," this factor was in Group 7, 90.0 per cent; Group

8, 95.8 per cent; Group 9, 96.0 per cent; Group 10, 100 per cent; Group 11, 100 per cent; Group 12, 84.6 per cent; an average for this group of 94.4 per cent.

The third column in each of the groups of three factors indicates the "present susceptibility" to dental caries. This was determined by the absence of active caries. For Group 1, this factor was 17.0 per cent; Group 2, 27.0 per cent; Group 3, 6.6 per cent; Group 4, 16.2 per cent; Group 5, 5.0 per cent; and Group 6, 0.0 per cent; that is, no teeth with active caries were found in this last group. The average for the series was 11.9 per cent. In the districts with high dental caries, the figures were: Group 7, 66.0 per cent; Group 8, 78.9 per cent; Group 9, 68 per cent; Group 10, 100 per cent; Group 11, 100 per cent; and Group 12, 84.6 per

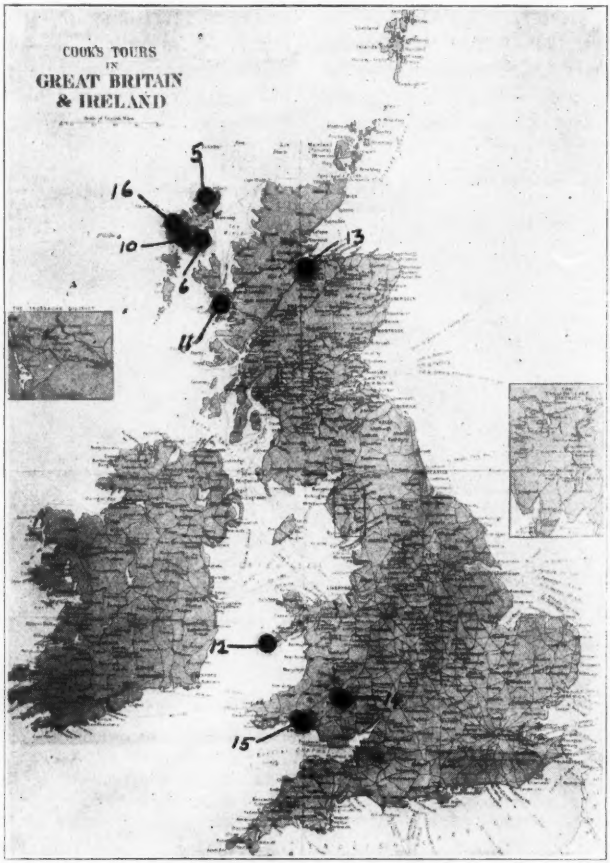

Fig. 15.—Districts in the British Isles where special field studies were made of incidence of dental caries: *5*, Lewis; *6*, Kyle of Scalpay; *10*, Tarbert; *11*, Airth of Sleat; *12*, Bardsey.

cent. The average for the series was 82.9 per cent.

An important phase of the special field studies conducted the summer of 1932 has included the securing of samples of saliva for chemical analysis. For this, 171 specimens were shipped or carried to my laboratories for study. In most cases, cultures were made in a special medium in order to study the incidence of the acidophilic organisms. This bacterial phase will be discussed later. Among the factors determined are total calcium, inorganic phosphorus, total phosphorus, solubility of the saliva for calcium, solubility for phosphorus and the ratio of calcium to inorganic phosphorus. The factor with which we are primarily concerned in this discussion is the behavior of the salivas when powdered bone chips are added to them. This is shown for inorganic phosphorus in Figure 12, above. In this connection it should be noted that while summer conditions in many districts on the whole are more favorable than winter, in some other districts they are distinctly less favorable. There are reasons that conditions were distinctly unfavorable at the time the saliva samples were taken. In high Alpine Valleys where they are dependent on dairy products as one of two principal sources of nutrition, they may suffer shortage because, in the summer, the dairy cows, and in some places also the goats, are sent to the high ranges to permit the harvesting to be done without their presence in the community (which is largely without fences), and, further, to permit the animals to have the advantage of the superior qualities of pasturing near the snow line; in other words, on the rapidly growing young plant life, which is in a very clear atmosphere, rich in ultraviolet rays, the conditions being responsible for the characteristic abundance and brilliancy of the flora of those regions. These valuable nutritive factors are placed in the milk by the cows. The milk in turn is made into cheese containing all the butter-fat and this cheese is stored for winter use. In the absence of the cows in summer, the nutrition of the children is greatly handicapped. Favorable results are obtained in spite of this disadvantage relating to the time the saliva samples were taken.

I have presented data indicating that the condition of immunity to dental caries is associated with physicochemical states of the saliva in which the inorganic phosphorus of the saliva passes from the saliva to the bone chips and that, in the condition of loss of immunity to dental caries, the movement is in the opposite direction; namely, from the bone chips to the saliva. One of the purposes of the different expeditions this summer was to obtain samples of saliva for checking these factors. To the left in Figure 12 are shown six groups with high immunity to dental caries, only 3.4 per cent of the teeth being affected in these six districts. Above is indicated the movement of the inorganic phosphorus in the saliva samples obtained as representative of these groups and all are shown to move downward in the direction indicating a movement out of the saliva to the bone chips. In the six groups on the right in which 25 per cent of all the teeth studied were now carious or had been so, the movement of the inorganic phosphorus is shown to be upward in the opposite direction; namely, from the bone chips to the saliva. At the time of this dictation, the chemical analysis of these saliva samples is still in progress and a further report will be presented later dealing with the behavior of the calcium and other chemical factors.

While it is impractical to make esti-

mations on the saliva chemical factors of the rats, a study of their blood has afforded important data as presented in Figures 4, 5, 6, 7 and 8. In Figure 10, the incidence of dental caries in the rats of the different groups has been shown on the basis of the average number of cavities per rat in each group. These show highest incidence in Groups 1, 2, 3, 7, 8, 10, 11 and 12. If reference will now be made to Figure 6, showing the percentage change of inorganic phosphorus plus bone, it will be observed that, in Group 1, 2 and 3, the inorganic phosphorus of the blood moved in the wrong direction, namely from the bone chips to the blood serum, as indicated by the crosses instead of the solid lines. In Group 7, this factor was reduced to zero. In Group 11, it was also in reverse phase, and in Group 12, reduced to zero. For those who are concerned with the changes in the blood which are likely to be responsible for the changes in the saliva, this will doubtless be significant.

An important phase of the collection of data this summer included the making of a record of the principal factors of nutrition in the various localities, the form in which the food was eaten, the obtaining of samples for chemical analysis and the ascertaining as nearly as possible of the percentage of certain foods used in the diet. Since cereals are, in all of these places, found to be an important factor, the particular cereals used and the form in which they were used were given detailed attention. One of the principal differences in the form in which cereals were used consisted of the method of preparation. In some districts studied, the cereals were made into porridges and breadstuffs from powdered whole grains. In some of these, small quantities of modified grain meals were in use, this usage extending to white flour and even bleached white flour. In some districts, practically all white flour was used. The proportion of these two for each place was studied and data were recorded. When the individuals for all the groups are arranged in progressive order on the basis of the ratio of white grain breads and its dilution with white flour to 100 per cent white flour and with the incidence of dental caries plotted with these percentages, we have the information revealed in Figure 13. It will be seen that in the individuals, regardless of the group and regardless of the other factors entering into the nutrition, but who were using all the minerals of the grain, the number of cavities as shown at the left was 0.3 cavity per person. When this whole grain bread was diluted with 25 per cent white flour, the cavities increased to 1.5 cavities per person. With the use of 50 per cent each of whole grain breads and white flour breads, the data reveal that there were about 2.3 cavities per person. With further increases in the proportion of white flour, the curve of incidence of dental caries goes up more rapidly, so that at 75 per cent white flour, there were 5.3 cavities per person, and with 100 per cent white flour breads, the data revealed seven cavities per person. When we use the data obtained by an analysis of a number of whole wheats and flours, we should find the phosphorus factor of the cereal at about 0.9 per cent in the whole grain breads and about 0.2 per cent in the white flour products. If we use the data provided in the table in Figure 2, we find that these figures are about 0.97 for the whole grain meal and 0.16 for white flour. The latter curve is shown in the continuous line. The phosphorus content of the different cereals and products is shown at the left.

Even without the data obtained from a quite detailed study of the diets of the

people living in districts of immunity and of susceptibility, it becomes possible to interpret the factors in these phenomena. Since body building and repair require an adequate amount of foods to be ingested which carry with them minerals and other chemicals including the activators, sufficient to meet the body's demands, and since Nature has constructed seeds of plants so that they provide these various factors, any nutritional program which seriously modifies these balances becomes deficient. This relates equally to minerals and activators, including the known vitamins. When the individuals in the high valleys, which formerly provided immunity, were satisfying all of their hunger demands for various stages and conditions of life by eating foods very high in both minerals and activators, conditions were provided for both good growth and a good defense, against natural or unnatural overloads. Where less efficient foods are provided such as prevail in modern procedures of preparing and selecting foodstuffs, the factor of safety is so greatly reduced that these people living under primitive conditions rapidly change physically and dental caries and other degenerative processes develop. Specifically, the change in the nature of the cereal factor of their foodstuffs, the total quantity of dairy products eaten and the reduction of the butter-fat intake in each of the general regions studied have apparently been largely responsible for the change from immunity to susceptibility to dental caries, and this handicap has not been compensated for by the increase in the variety of foods, including marked increase in the availability of fruits and vegetables. In the districts where immunity to dental caries existed not only for the present generation examined, but also for preceding centuries, there was also a marked absence of irregularity of the teeth. On the contrary, those areas with high susceptibility, or with even recently acquired loss of immunity, showed much deformity of the arches and irregularity of the teeth, expressing itself in even a single generation and indicating that the stress existed all through the developmental and growth periods.

COMMENT

An interpretation of the various factors in order that they may be applied to the districts in which field studies were made requires that some further data be in mind. In the high Alpine valleys, rye constitutes the principal locally produced cereal. Usually, the only other grain that is grown is barley, and it is very limited in use in comparison with the rye. In the Outer Hebrides, practically no rye or wheat is locally produced, the locally grown cereal being very largely oats with a small amount of barley. Wheat does not readily ripen in either of the above mentioned places. In the Island of Bardsey, wheat is grown easily, as are also rye and oats. Corn was not an important factor of diet in any of the communities included in this study. Where wheat was imported, it was very largely imported as white flour. It is also important to note that white flour was available in every place where transportation facilities made its shipment easily possible. The only areas found where white flour was not easily available either as flour or as white bread and white flour products were places where it could not readily be delivered. It was frequently possible to go from an area of very high immunity to dental caries to one of very low immunity in one hour or, at the most in a few hours. In Switzerland this usually meant climbing down from a place of difficult access to one more accessible.

The geographic positions of the places studied are presented in Figure 14, the white pins indicating places from which I have been receiving butter samples during the past year, usually every two weeks, for analysis of the vitamin levels. The black pins indicate the districts in Switzerland which were studied last summer. In order to check the rôle of altitude, since the districts of immunity were all found from about 4,000 to about 6,000 feet, care was taken to study places with comparable conditions. It is of interest that Ferdon, Kippel, Wiler, Blatten, Ayer, Grachen and Visperminen, which were places with high immunity or a low incidence of dental caries, are all situated in high districts in the Alps. In none of these places did we find evidence of a program of prophylaxis or any evidence of oral hygiene. Indeed, it is doubtful that many of the people studied in these districts even owned or ever saw a toothbrush. St. Moritz represents a comparable climate and altitude to all of the places just mentioned. Its fame is world wide as a health resort, both winter and summer. Every child studied in St. Moritz showed evidence of an efficient program of prophylaxis and all had been taught oral hygiene in the public schools, yet almost every child had suffered severely from dental caries at some time and 78.9 per cent had active dental caries at the time of the examination. Examination revealed that 29.8 per cent of all teeth had been attacked by dental caries. This, of course, occurred in spite of an excellent prophylactic program. Similarly, isolated districts in the Isle of Lewis, in the Outer Hebrides, and in Kyle of Scalpay in the Isle of Harris are shown to have a very low incidence of dental caries. These are at sea level. There was little if any evidence that oral hygiene had ever been taught or practiced. The locations of the places studied in the Outer and Inner Hebrides and other parts of the British Isles are shown in Figure 15, by the dark pins.

As we proceeded in Switzerland to Herisau, a thriving modern city in the plains district near Lake Constance in the northeastern part of Switzerland, we again found boys and girls with much evidence of home care and careful teaching in methods of oral hygiene; yet 24.7 per cent of all teeth showed caries; 89 per cent of individuals had had dental caries, and 68 per cent had active dental caries at the time of the examination. This is most significant as a commentary on the inefficiency of prophylaxis as a means of control of dental caries.

The data here presented relative to the kind and form of cereals used indicate strongly that it does not matter so much what the kind of cereal used or the form in which it is used, provided all of the natural mineral content of that cereal is made available. This is also emphasized in the group of studies presented under "Results from Feeding Fifteen Different Diets to Fifteen Groups of Rats." The question of the form of the units of food that are taken, whether soft or hard, coarse or fine, does not seem to have been the determining factor. In the series obtaining the whole corn, there was a higher percentage of cavities than in either of those groups receiving the fine or coarse corn. Those receiving the fine corn had more cavities than those receiving coarse corn. This is not in accordance with the data presented by Hoppert, Webber and Canniff. I would emphasize that the fine feed corn group (2) and the fine seed corn group (12) each had more cavities than the coarse feed corn group (3) and the coarse seed corn group (11). Since none

of these had so many cavities as Group 1, receiving both the fine and coarse corn, though all had liberal caries, it is suggested that we should not put too much emphasis on the coarseness or fineness of the food.

It is important to note, particularly for those who would put great emphasis on the hardness of the food eaten as a means of muscular exertion for the development of the bones of the face, that the people living in the high Alpine districts that were found to have a high immunity to dental caries were eating a hard bread usually weeks old, sometimes several months old and dried out until it required much effort for its mastication, while the people in the Outer Hebrides who also were found to have high immunity to dental caries were using a large part of their cereal in the form of soft porridge. True, the oat cake is crisp, but it is thin and relatively more easily masticated than the Swiss dark whole grain bread. The evidence does not support the view that the texture of the food is a determining factor.

The matter of the availability of the various vitamins has been studied and further data will later be presented. Some factors can be determined readily from a study of local conditions. In none of the immune districts included in this study were citrus fruits available. Many of the immune individuals in both the high Alpine valleys and the interior of the Isle of Lewis have probably never eaten an orange or grapefruit and in neither district were wild berries provided as a native food. Some citrus fruits come into Switzerland from Italy and Spain, but they reach only communities provided with adequate means of transportation. No district was found with immunity where imported fruits were available. This would seem at once to exclude the possibility of vitamin C being a controlling factor in these districts. The inhabitants of Vispertiminen had vineyards down the mountainside. Since there are many places where immunity to dental caries existed a few years ago and where that immunity has been lost coincidentally with the opening up of means of easy transportation of foodstuffs, it has not, of course, been the introduction of citrus fruits to formerly immune areas which has contributed to the loss of immunity. It is also significant that in no place was immunity to dental caries found where there was not a bountiful source of fat-soluble vitamins in a natural food product, which my investigations have demonstrated to constitute a principal controlling factor in dental caries in many individuals and in many districts. For the Inner and Outer Hebrides, fish products are available and are used liberally. A particularly favored dish in the Isle of Lewis consisted of cod's head stuffed with a mixture of oat meal and chopped cod livers and baked. Heads with large muscles were selected and the brains left in position. This would furnish vitamin D and phosphorus besides the minerals in a readily available form. Of course, no fish were available for providing vitamins in the isolated high Alpine valleys. Probably many of those individuals have never seen a fish. Their source of fat-soluble vitamins is primarily from the dairy products used as milk, cheese and butter. The cheese contains the full butter-fat. With the introduction of contact with the outside world by means of transportation, easy access is had to markets for dairy products. These markets are generally utilized as a means of obtaining ready money for the purchase of machine made clothing and canned foods and flour. In the Hebrides, the thatched roof that covers the family covers the cows and goats. The walls were built of

stone and many of the homes were without windows and chimneys. The thatched roof has two purposes, protection from the weather and the preparing of a special fertilizer for the soil through the burning of the peat for most of the year. The few trees on the Isle of Lewis have been planted in chiefly one place, near the coast. The island is largely covered with peat from a few inches to 20 feet in depth, except for the areas covered with bald rocks. The geologic form is granite of the oldest type. The mineral calcium is very scarce. The plant that is seen most everywhere is purple heather, as is also true in much of north Scotland. About the only animals on the island are the domesticated blackface sheep and a few highland cattle or mixed breeds of highland with others. These animals are very hardy and can live out through the winter. The little pasture grass that these animals can pick up enables them to collect the plant calcium and phosphorus and make these minerals available in the very limited dairy products which were part of the former primitive diet and still is for a few people living in a primitive manner. Eggs are also used. With the introduction of modern foods in condensed form and generally low in minerals, this important source of nutrition is rapidly dwindling in the districts having contact with commerce, with the result that passing from an immune area to the principal seaport of Stornoway, out of 100 people between the ages of 16 and 35 (estimated), there were found twenty-five wearing artificial teeth, and about an equal number showed that they had lost many teeth or parts of them from caries. All the people speak the same language, Gaelic. Some of them speak English. The breakdown was so rapid and clear cut that people from 50 to 80 years of age still had most or all of their teeth without evidence of active caries. These people were insisting on having their original diet. The younger generation which had access to the modern diet had rampant dental caries. In some districts, 60 per cent of the people between 20 and 60 years of age were wearing artificial dentures, often both upper and lower. This condition of increase of dental caries was reported in many districts as being progressively more severe. An analysis of these diets and of living conditions will be presented in another communication.

In further consideration of the rôle of activating substances, including the known vitamins, important data are provided in Figure 9. In this, fifteen diets were used, with the same basic ration for all, the modification being chiefly in the kind or form of the cereal factor. A glance at the chart reveals at once that dental caries was particularly high in Groups 1 and 2, moderate in Group 3 and completely absent in Group 4, who received wheat instead of corn; also absent in Group 5, who received fine and coarse feed corn as provided to Group 1 plus all they wished to eat of high vitamin butter. In Group 6, receiving the same as Group 1 plus 1 per cent high vitamin butter and 1 per cent high vitamin cod liver oil, there were no gross cavities but several small ones. In group 7 receiving the same as Group 6 plus wheat germ, there were moderate carious cavities. In Group 8, receiving the diet of Group 1 plus wheat germ, there was moderate caries. In Group 9, receiving the wheat of Group 4 plus wheat germ, there was no caries. In Group 10 receiving the diet of Group 1 plus dicalcium phosphate in an inorganic form, there was considerable caries. In Group 11, receiving coarse siftings of a seed corn differing from that employed

in Group 1 only in kind of corn, there was considerable caries. Group 12, receiving the diet of Group 1 plus the fine siftings of seed corn, had very active caries. Group 13, receiving the Red Cross whole wheat, had no caries; while those in Group 14, on Red Cross flour, had much caries. This group received not only less minerals but also less of the activators that would have been provided in the wheat embryo. Group 15, receiving the Red Cross bran and middlings mixture, had no caries. It is at once seen that something was provided in the butter that was made available with the diet in Group 5, which otherwise corresponded with Group 1, and which was capable of changing that diet so that it provided complete immunity to dental caries. The very small amount of butter and cod liver oil furnished to Group 6 reduced caries to a very low level. That the activators provided in wheat germ are more efficient when used to supplement wheat rather than to supplement corn is suggested by the complete immunity of Group 9 as compared with Group 8.

An important new light on the relation of the phosporus of the blood to dental caries susceptibility and immunity is provided by shaking powdered bone into the blood serum and noting the change it produces in the level of inorganic phosphorus, which is shown in Figure 9 to follow the caries susceptibility for these different groups.

If we review the summaries of accumulated facts as provided by Arthur D. Black[2] and Robert Kesel,[3] we note that the evidence stresses the necessity that caries shall begin on the outer surface of the tooth and then usually only in protected areas, never involving deeper structures until the external surfaces had been penetrated and that it is initiated and accompanied by a decalcifying process in which acid-producing microorganisms seem to have a part. These phenomena are apparently not related to acidity of saliva, but are related to its buffer capacity. All of these conditions could be provided by variation in physicochemical characteristics of the fluid bathing the external surfaces of the tooth, provided it could be shown that it contained forces which could progressively carry minerals in most minute form either into the tooth surface by absorption or out of the tooth surface in accordance with changes in that fluid. The information given here of an increase or decrease in the inorganic phosphorus of the saliva in accordance with whether that individual was found to show immunity or susceptibility to dental caries is presented in support of this premise. The data provided in the illustration of the teeth before and after treatment (Fig. 10) show the difference in the penetration of silver nitrate and Figure 11 shows the actual building of new tooth structure when the blood and saliva are put in such condition that minerals are deposited rather than taken up. Many additional data are available on this point. That this change in the chemical aspect of the saliva can be produced by nutritional changes is supported by the evidence presented here and in former communications as reviewed in the introduction. When these interpretations are applied to the review of fundamentals as presented by Kesel, we find why both ancient and modern primitive people living in their native environment develop little decay. We find supporting evidence in these studies of districts of immunity or susceptibility. When we apply these principles to the phenomena associated with periods of physical stress, the active growth of childhood and adolescence,

pregnancy and disease, we find much evidence that the need of the body for the minerals and activators with which body tissues are built and repaired incident to those overloads prevents the development of a normal saliva for the environment of the tooth. The reason that decay of teeth ceases even though the bacteria are still present and even though culture mediums suitable for their needs are provided now becomes apparent. We also see why crowns of teeth when mounted on artificial dentures and worn in the mouth can also be attacked by dental caries as though in their normal position. When we apply this view to the areas of susceptibility which have been indicated to be vulnerable areas owing to the collection of foreign material in any location where the saliva cannot, particularly when its factor of safety is low, maintain a margin of safety, because the physical environment prevents its ready access, we see why saliva with a low but positive factor of safety can maintain immunity on surfaces that are liberally exposed. This provides an explanation for the observation that small proximal cavities or caries under fillings may progress; whereas, if the cavity is opened so that saliva has free access, so long as its margin of safety, even though small, exists, caries is inhibited. In the protected areas, a very high state of defense in the saliva would have to be provided in order to maintain immunity. Further, when we consider the problem of the progressive softening of teeth in cases of rampant caries, as in pregnancy and some other conditions and later the hardening of these teeth, we find not only an adequate explanation, but also a physical condition which we have demonstrated on teeth removed from the mouth and which shows not only change in structure on the basis of hardness, but also a change in chemical content varying in amount and in chemical expression. In most cases, a greater reduction in phosphorus than in calcium occurs. In some cases, there is a reduction in calcium. In a later communication, these data will be presented and will be shown to have a direct relationship to the nutritional deficiency and also to the nature of the local expression in both the teeth and their supporting structures.

SUMMARY AND CONCLUSIONS

1. Supporting data are presented for a new interpretation for the phenomena of dental caries based upon a physicochemical state of the saliva which provides and largely controls the immediate environment of the tooth.

2. These new interpretations have been supported by data provided from the study of both experimental animals and human beings, the latter living under both primitive and modern social conditions.

3. The specific requirements indicated by past investigations regarding the etiology of dental caries can be largely if not completely explained on the basis of these physicochemical qualities of the saliva.

4. People living under modern conditions which provide either immunity or susceptibility reveal physicochemical conditions of the saliva apparently competent to create the clinical dental conditions found in those individuals.

5. A new theory for the phenomena of dental caries has been provided supported by clinical data which furnish the basis for a program shown to be competent not only to prevent the development of dental caries but also to control it after it is established.

6. Data are presented which provide an explanation not only for the general

immunity of wild animals from dental caries but also for the immunity of primitive peoples, both ancient and modern.

7. Light is thrown on many degenerative processes of both hard and soft tissues also resulting from the accumulated effects of faulty nutrition and means are indicated for the reduction of their severity and incidence.

IS TRAUMATIC STRESS PER SE A CAUSE OF HYPERCEMENTOSIS?*

By JAMES L. ZEMSKY, D.D.S., New York City

IN THESE days when the medical and dental professions are continually confronted with numerous perplexing problems, the solution of which demands concentration of all our efforts, a presentation of the relationship between hypercementosis and traumatic stress may appear trivial; and yet, after reflection, this apparently insignificant subject will assume sufficient importance to warrant its careful consideration. As a clinician whose practice is confined to oral surgery, diagnosis and consultation, I am called on, in private practice as well as in hospital work, not only to treat seriously sick patients, but also to render opinions and advice to those enjoying good health who have been in the habit of having periodic physical examinations, including careful roentgenographic surveys of the mouth. Not infrequently, these have disclosed conditions which, although not pathologic, cannot be regarded as normal. Among them, unerupted, impacted and malposed teeth, areas of sclerotic bone, deeply buried root apices with no evidence of any disturbance and hypercementosed teeth have been quite prevalent. Similar conditions may be observed in the mouths of those suffering from various systemic disturbances. In many instances, it has

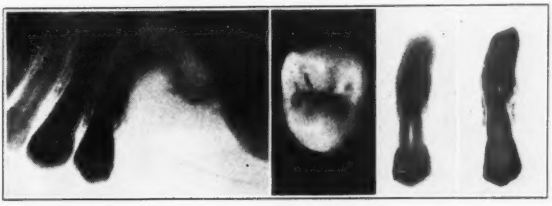

Fig. 1.—Maxillary first bicuspid in woman, aged 27; a vital tooth presenting no sign of traumatic stress and yet showing a marked degree of hypercementosis.

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been rather difficult to determine definitely the nature of the advice to be given to these various patients. In order to find a guide to facilitate this task, I turned to the research laboratory, and discouraged by failure to receive enlightenment from that source, returned to the clinic for my investigation.

Fig. 2.—Enlarged section of tooth shown in Figure 1.

DEFINITION OF HYPERCEMENTOSIS AND SCOPE OF THIS PAPER

For clarity, it is necessary to define the term "hypercementosis." The definition given in Brubaker's[1] textbook is fairly accurate. It describes hypercementosis as "a secondary deposit or an increase of volume of the cementum of a tooth beyond the normal limit." Since it is difficult to determine clinically the normal limit of cementum, this definition should be modified to read: "Hypercementosis is a secondary deposit of

1. Brubaker, A. P.: American System of Dentistry, New York City, Dental Items of Interest Publishing Co., 1887.

Fig. 3.—Lower first bicuspid in man, aged 29. The tooth is in buccal eruption, presenting no evidence of traumatic stress. There is a marked degree of hypercementosis.

Fig. 4.—Enlarged section of tooth shown in Figure 3.

cementum beyond the normal limit to such an extent that it is readily discernible by an unaided ocular examination." This makes a diagnosis of hypercementosis with the aid of the roentgen rays both simple and definite.

Hypercementosis is generally regarded as a result of traumatic stress and of infection, producing a low grade irritation in the periodontal tissue which eventually gives rise to the condition. Whether it is traumatic stress or infection that is responsible for hypercementosis, or whether other etiologic factors are involved, is not the subject of this paper. Bunting[2] mentions pyorrhea and periapical infection among the etiologic factors. This may be true, and there may be others. A discussion of these theories is beyond the scope of this paper, which is confined to a consideration of whether traumatic stress in itself is a direct cause of hypercementosis.

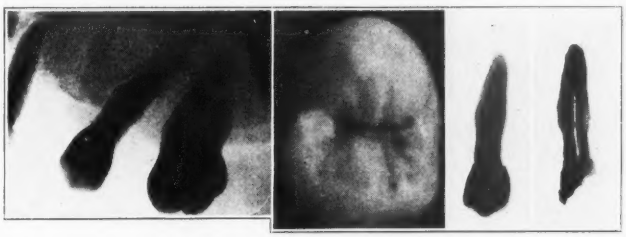

Fig. 5.—Maxillary second bicuspid (patient, aged 36). The tooth was not subjected to traumatic stress, as is evidenced from the occlusal surface. There is a marked degree of hypercementosis present.

TRAUMATIC STRESS AND HYPERCEMENTOSIS

Hypercementosis may be observed on all teeth, vital or pulpless, regardless of whether the root canals have been treated and filled or not. It is seen on normally erupted as well as on partially erupted and unerupted teeth. These may be merely malposed or impacted. It is found on teeth in perfect occlusion as well as on those completely out of occlusion, including teeth in so-called traumatic occlusion.

There is a general impression among those of our profession that hypercementosis is a result of traumatic stress.

A brief reference to the literature, old as well as recent, will show clearly that traumatic force is considered responsible for the production of hypercementosis. Of the old textbooks, Brubaker's[1] should be mentioned, and of the newer publications those of Pollia[3] and Bunting[2] are important.

Pollia's[3] discussion of the relationship between traumatic stress and hypercementosis includes a presentation of several roentgenograms which are described as follows:

Fig. 271. This is a hyperplasia within the peridental lamella or excementosis. It is a picture of the lower left bicuspid area. The bicuspid is not in focus. *The first bicuspid has evidence of traumatic stress,* as is indicated by the sharp lines of the occlusal surface image. Examination of the root reveals

2. Bunting, R. W.: Textbook of Oral Pathology, Philadelphia, Lea and Febiger, 1929.

3. Pollia, J. A.: "Alveolo-dental Radiology," New York City, Dental Items of Interest Publishing Co., 1930.

the presence of three types of radiopacity; namely, the dark lineal radiolucence representing the root canal; peripheral to this is the lighter radiopacity, or increased illumination, which is normal root structure composed of dentin and *immediately around it lies the pathological (?) cementum that has a slightly less radiopacity.*

This, that Pollia calls pathological cementum is nothing but hypercementosis. Referring to "Fig. 272" he continues:

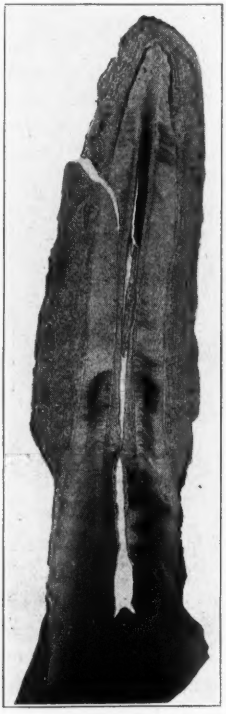

Fig. 6.—Enlarged section of tooth shown in Figure 5.

This radiograph shows the presence of excementosis, or hyperplasia, affecting the tissues within the peridental lamella of the cuspid and second bicuspid in the same subject and on the same side. *The cuspid and bicuspid are victims of traumatic occlusion, as is indicated by the wear of the occlusal surfaces.*

Discussing the etiology of hypercementosis, Bunting[2] refers to a low-grade irritation in the periodontal tissues which results in hypercementosis. In enumerating the causes of hypercementosis, Bunting first mentions occlusal traumatism.

It is quite natural, therefore, that the rank and file of the profession should hold this view. I had an opportunity to ascertain this in connection with an investigation that was conducted some time ago by the Research Department of Oral Histology and Embryology of Columbia University. In an effort to determine the clinical significance of hypercementosis, a questionnaire was sent to members of the profession, and, from the replies received, it was evident that traumatism was regarded as a cause of this condition.

My observations do not support this view. A considerable number of cases were studied, but no causal relationship between hypercementosis and traumatic force could be established. On the contrary, the results of my observations indicate that traumatism in itself, barring other conditions, does not produce hypercementosis. This conclusion is based upon the premise that if trauma produces hypercementosis, the condition should be demonstrable on most of the teeth thus affected, which, as may be seen from the accompanying illustrations, is not the case. With illustrations showing teeth exposed to various traumatic forces and presenting different degrees of hypercementosis will be shown others of teeth exposed to the same types of traumatic stress and yet presenting no signs of hypercementosis. The deductions from such observations are obvious. Traumatic stress *per*

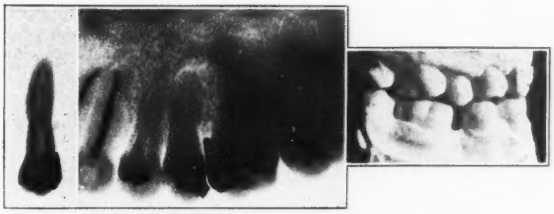

Fig. 7.—Casts of teeth of patient, aged 26, showing maxillary second bicuspid out of occlusion. The roentgenogram reveals that the tooth is hypercementosed, as is also shown clearly in the roentgenogram of the extracted tooth. This hypercementosis could hardly be caused by traumatic stress.

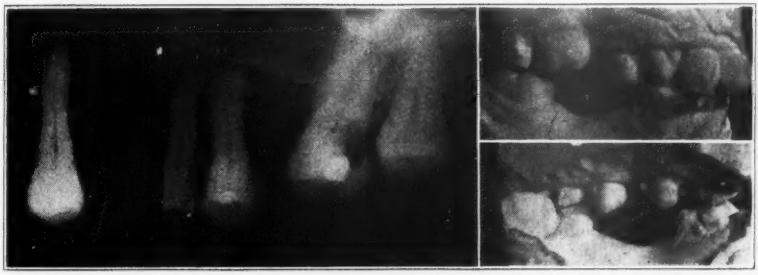

Fig. 8.—Casts of a maxillary second bicuspid in woman, aged 40, presenting hypercementosed maxillary second bicuspid which is out of occlusion.

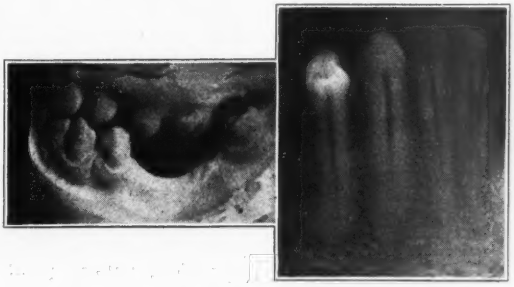

Fig. 9.—Cast and roentgenogram of mandibular first bicuspid. The tooth is out of occlusion and presents a marked degree of hypercementosis.

se is not a cause of hypercementosis. Such a demonstration may not be acceptable at present as a conclusive proof, but it certainly is convincing.

This is another type of research problem which demands further clinical investigation by the general practitioner, as previously called to the attention of

the profession.[4] The task is relatively simple. On the one hand, traumatized teeth should be roentgenographed and the presence or absence of hypercementosis thus determined. On the other, when roentgenograms reveal hypercementosis, the presence or absence of traumatic forces should be ascertained and the relationship thus established.

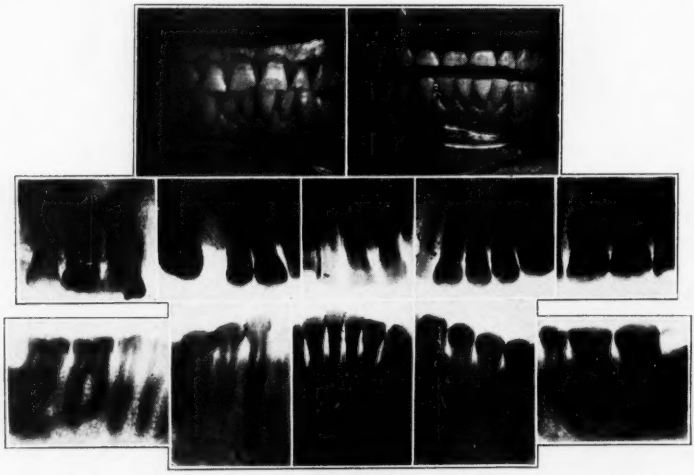

Fig. 10.—Tip to tip bite in man, aged 35. The teeth are ground down and show a considerable degree of hypercementosis. (Compare Fig. 11.)

Fig. 11.—Tip to tip bite in patient aged 69. The teeth are markedly worn down. Despite the excessive occlusal stress and the age of the patient, there is no sign of any hypercementosis. (Compare Fig. 10.)

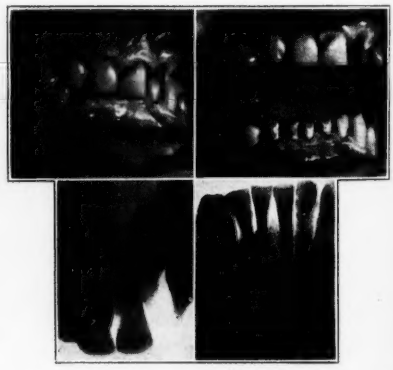

Fig. 12.—Teeth of patient, aged 51, ground down owing to excessive occlusal stress. Despite traumatism, there is no sign of hypercementosis.

4. Zemsky, J. L.: Clinical Research by General Practitioner: New Concept of Dental Research with Plan for Achievement, J.A.D.A., 19:12-28 (January) 1932. Zemsky, J. L., and Bödecker, C. F.: Scientific Investigation for General Practitioner of Dentistry, ibid., 19:935-949 (June) 1932.

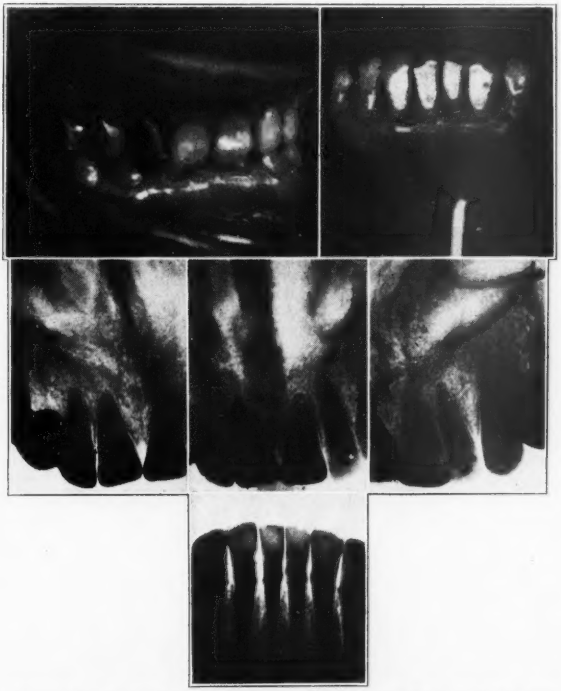

Fig. 13.—Deep overbite in patient, aged 39, producing marked occlusal trauma. There is no hypercementosis revealed by the roentgenograms.

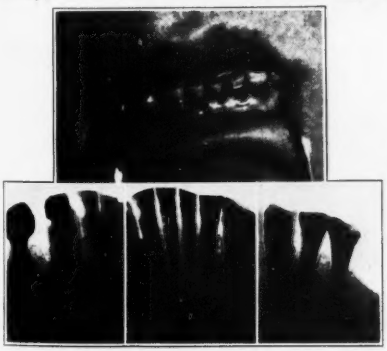

Fig. 14.—Case of man, aged 65, presenting nine teeth in mandible. The patient had been wearing an upper denture for more than thirty years. The teeth are traumatized, as evidenced by the wear of the crowns. There is a considerable degree of hypercementosis.

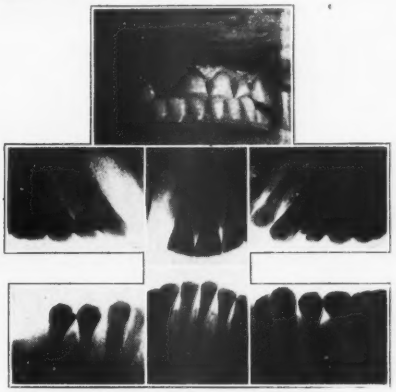

Fig. 15.—Protrusive bite in patient, aged 43. Despite the marked occlusal stress which has traumatized the teeth, no hypercementosis is discernible.

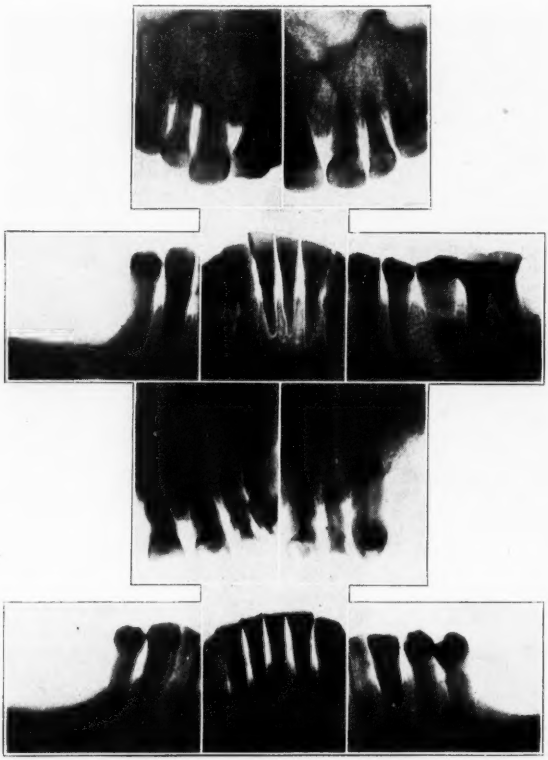

Fig. 16.—Maxillary and mandibular teeth showing signs of marked traumatic stress but presenting no hypercementosis. (Compare Fig. 19.)

Fig. 17.—Maxillary central and lateral incisor and bicuspids showing no signs of traumatic stress and presenting various degrees of hypercementosis.

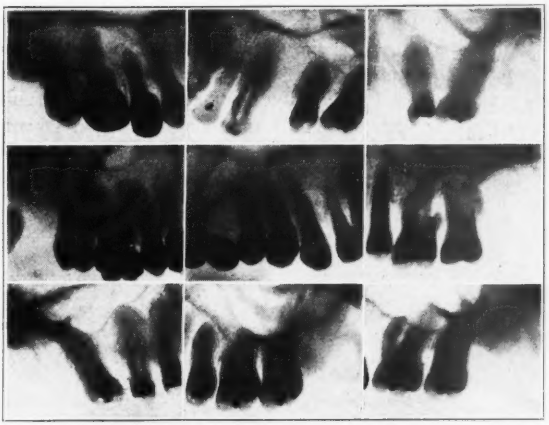

Fig. 18.—Maxillary posterior teeth showing no evidence of traumatic stress, but presenting hypercementosis.

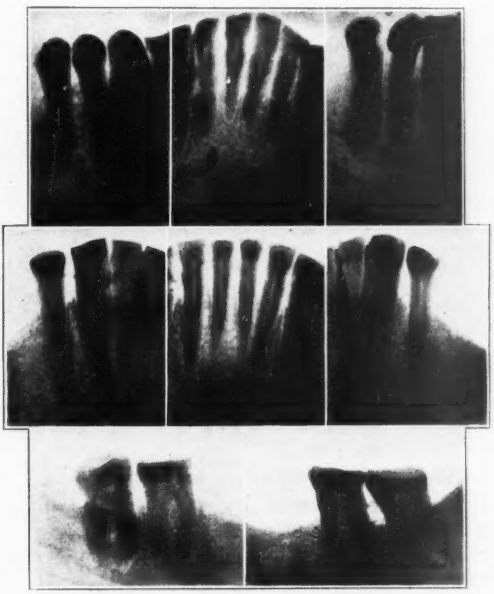

Fig. 19.—Mandibular anterior and posterior teeth showing marked signs of traumatic stress. All these teeth are hypercementosed.

REPORTS OF CASES AND FINDINGS

The following cases will demonstrate the points brought out in the foregoing discussion. The patients studied were male and female and of various ages. Their physical organisms differed, and their social stratum and economic status were likewise diverse. The series of patients whose photographs and roentgenograms are described present teeth subjected to traumatic stress. Here, there may be observed the tip to tip bite resulting in marked grinding down of the crowns. Patients with the deep overbite and protrusive bite may also be seen. Some show the presence of hypercementosis; others do not.

Among the cases here presented, there are those which show certain teeth out of occlusion, but which nevertheless present hypercementosis. As a further argument in support of the idea that traumatism is not responsible for the production of hypercementosis, attention is directed to those partially erupted and malposed teeth which cannot be regarded as affected by traumatic force, and to others which, despite this, are not affected by hypercementosis. The few roentgenograms showing the presence of calculi, but presenting no hypercementosis, a condition readily observed by every practitioner, should prove sufficiently convincing to refute the idea that traumatism due to calculi produces hypercementosis.

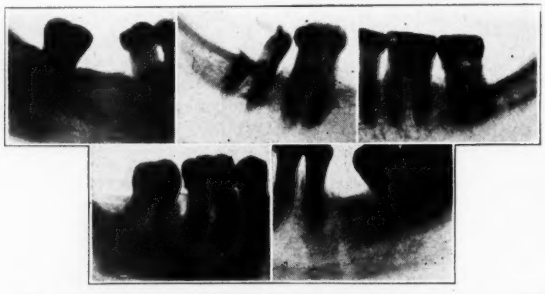

Fig. 20.—Mandibular posterior teeth subjected to traumatic stress. All the teeth are hypercementosed.

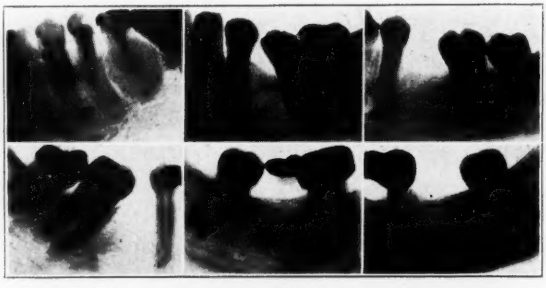

Fig. 21.—Mandibular posterior teeth showing no signs of traumatic stress, but presenting marked degree of hypercementosis.

The cases here presented demonstrate that traumatism, no matter in what form, is not responsible for hypercementosis.

SUMMARY

It is maintained that traumatic stress in one form or another produces hypercementosis. This is obviously based on the fact that hypercementosis has been observed on all types of teeth which give evidence of having been affected by traumatic force. On the other hand, a large percentage of unerupted, impacted and malposed teeth show no evidence of hypercementosis, which is not infrequently seen on teeth giving no evidence of occlusal trauma. There are likewise many teeth completely out of occlusion which show hypercementosis, such as malposed and partially erupted and impacted teeth. On the other hand, teeth with very marked signs of traumatic occlusion present no hypercementosis. What conclusions are to be drawn in view of these observations? Is it not justifiable to doubt the theory of many writers and other members of our profession which attributes the production of hypercementosis to traumatism?

If traumatism produces hypercementosis, as indicated by specimens of teeth showing signs of both traumatism and hypercementosis, it is but logical to expect to find the existence of hypercementosis in cases in which traumatism affects a tooth. But this is not so, as is sufficiently illustrated both in roentgenograms of teeth subjected to traumatic stress and in extracted hypercementosed teeth. Moreover, if traumatism is the cause of hypercementosis, it should be present in cases of hypercementosis. This is likewise contrary to our observation, since marked hypercementosis is seen on teeth not affected with traumatism. This is illustrated by photographs and roentgenograms of patients presenting hypercementosed teeth which are not only not traumatized but are also out of occlusion.

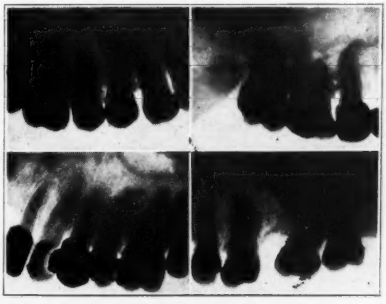

Fig. 22.—Maxillary posterior teeth with operative and prosthetic restoration. These teeth show hypercementosis.

CONCLUSIONS

A study of the existing relationship between hypercementosis and traumatism based on a considerable number of cases would seem to indicate definitely that:

1. There is a lack of direct or causal relationship between traumatism and hypercementosis.

2. The theory that hypercementosis is caused by traumatic stress *per se* not only does not seem to be well-founded, but, also from present observation, would appear to be refuted.

3. Traumatism *per se* is not responsible for hypercementosis, but further extensive clinical observation by the general practitioner with adequate checking of roentgenographic findings and clinical facts is necessary to prove this contention.

4. Under certain circumstances, traumatism may exert some influence conducive to the production of hypercementosis, such influence being beyond the scope of this paper but presenting a topic for further investigation.

5. All teeth affected by traumatism or presenting hypercementosis should be studied roentgenographically and the findings carefully recorded, so that conclusive proof of this problem may be adduced from the accumulated mass of material.

147 Fourth Avenue.

Fig. 23.—Mandibular anterior teeth with calculus and showing hypercementosis. (Compare Fig. 24.)

Fig. 24.—Mandibular anterior teeth with calculus, but showing no signs of hypercementosis. (Compare Fig. 23.)

AN EVALUATION OF THE VARIOUS REAGENTS USED IN THE TREATMENT OF THE SPIROCHETOSES*

By L. J. BELDING, M.D., and P. H. BELDING, D.D.S., Waucoma, Iowa

THE conception of the spirochetoses as a disease entity is of comparatively recent origin. Their unity was first satisfactorily demonstrated by Kritchevesky and Seguin[1] in 1924. Since then, the investigations of many workers throughout the world, particularly Castellani,[2] Tunnicliff,[3] Managabeira-Albernaz[4] and Smith,[5] have, experimentally and clinically, fully and completely substantiated the original observations and allowed for the irrevocable placement of the spirochetoses among the disease entities.

Fusospirochetal disease is generally initiated as a chronic gingivitis and, from their location in the gums, the specific organisms cause more pathologic buccopharyngeal conditions than any other group of bacteria. They are the chief group of organisms associated in the production of periodontal disease and oral foci of infection, and, at times, cause local and remote severe, and often fatal, diseases.

The significance of the fusospirochetes in their relationship to man may be better appreciated when it is considered that 95 per cent of all dentulous persons chronically harbor these organisms, localized subgingivally.

Davis[6] is of the opinion that these organisms may be found constantly in every mouth and throat as opportunists, and that, under suitable conditions, they may produce putrid and gangrenous lesions.

In order that a clearer understanding of fusospirochetal disease may be obtained, we quote from the introduction to Smith's[5] recent and authoritative book "Fuso-Spirochetal Disease."

> But they (fuso-spirochetes) are probably the most important member in a symbiotic group of anaerobic organisms which is capable of initiating a severe and often fatal disease. This group of anaerobes, which is composed of spirochetes, fusiform bacilli, vibrios and cocci, is surpassed only by *Treponema pallidum* and *Micobacterium tuberculosis* in the wide variety of acute and chronic lesions it produces in various parts of the body. Situated in the tonsils and about the teeth they are in a strategic position to initiate disease locally in the tonsils, gums, cheeks, tongue, pharynx and larynx, or to invade the eyes, ears or

*Read before the Staff Conference. U. S. Naval Hospital, Great Lakes, Ill., Nov. 23, 1932.

1. Kritchevesky, B., and Seguin, P.: D. Cosmos, 60: 781 (Sept.) 1918; 66: 511-522 (May) 1924.

2. Castellani, A.: Lancet, 1: 1384, 1906.

3. Tunnicliff, Ruth: J. Infect. Dis., 25: 132 (Aug.) 1919.

4. Managabeira-Albernaz, P.: Laryngoscope, 39: 1-15 (Jan.) 1929; Arch. Otolaryngol., 12: 49 (July) 1930.

5. Smith, D. T.: Oral Spirochetes and Related Organisms in Fuso-Spirochetal Disease, Baltimore: Williams and Wilkins Company, 1932.

6. Davis, D. J., in Jordan and Falk: Newer Knowledge of Bacteriology and Immunology, 1929, p. 655.

sinuses. They may be aspirated and infect the trachea, bronchi or lungs, or may be swallowed and cause disease in the esophagus, appendix or colon. By means of fingers contaminated with saliva these anaerobic organisms may be carried to the skin or to the external genitalia. Through emboli they may reach the brain, lungs or other internal organs. Finally, this is a preventable disease. The source of the infection is in diseased tonsils and in diseased gums, and if these foci are eliminated, then all the local and general infections with these anaerobic organisms might be prevented.

The organisms were probably among the "wretched beasties" observed in the seventeenth century by Leeuwenhoek.[7] He certainly must have seen the larger forms when he studied the gingivial exudate from the old man who never in his life had cleaned his teeth. The significance of these organisms pathologically was suspected in 1867 by Leyden[8] and Jaffe, but it was not until after the early part of this century that their capacities in that direction began to be generally recognized.

The morphology of the fusospirochetes was studied in 1882 by Miller,[9] who described them in a detailed, though somewhat unsatisfactory, manner. To date, their ecology, morphology and taxonomy remains in dispute.

Since Plaut,[10] in Germany, and Vincent,[11] in France, seriously called attention to the pathogenic potentialities of the fusospirochetal combination, interest in and respect for it has steadily increased, until, at the present time, it is regarded by many writers as the most important of the anaerobic symbionts.

We regret that we cannot enter into the controversial discussion as to the etiology of the buccopharyngeal diseases, but in a paper of this nature, we are limited to the allegation that all of the necrotic processes, with rare exceptions, are initiated by the fusospirochetes. We further allege that the presence of the fusospirochetal organisms in the mouth, particularly in the gingival sulcus, is pathognomonic of underlying disease which may be gross or microscopic, and is an indication for prophylactic or therapeutic measures.

Kritchevesky and Seguin[1] have classified the buccopharyngeal spirochetoses as follows: acute: noma, Vincent's angina, ulceromembranous stomatitis, mercurial stomatitis, etc.; chronic: chronic gingivitis, chronic fusospirochetal tonsillitis and pyorrhea.

For a proper understanding of the treatment of these conditions, it is essential that we say a few words about spirochetoses in general. The spirochetoses are a very large group of infectious processes caused by a group of organisms of which the spirochete is the most important member. Any part of the body may be attacked and the manifestations are extremely varied. They may be fulminating, producing death in forty-eight hours, or they may be latent and symptomless.

To the end that the frequency and protean manifestations of the spirochetoses may be appreciated, we offer the classification in Table 1.

It must be understood that this classification is relative, for from the local to the remote and the acute to the chronic is but a step, and is a reversible process.

Similarly to the streptococcal infections, any part of the body may be at-

7. De Kruif, Paul: Microbe Hunters, p. 18.

8. Leyden, E., and Jaffe, M.: Deutsche Arch. f. Klin. Med., 11: 488, 1867.

9. Miller, W. D.: Micro-Organisms of Human Mouth, Philadelphia: S. S. White Dental Mfg. Co., 1890.

10. Plaut, H. C.: Deutsche Med. Wchschr., 20: 920, 1894.

11. Vincent, H.: Arm. de l'Inst. Pasteur, 10: 488, 1896.

tacked and, like these infections, the spirochetoses exhibit a chemicomechanical predilection for certain tissues and, in these sites, disease is produced sufficiently often and the symptoms and clinical-pathologic findings are so nearly constant that these processes have been considered as distinct entities. Among the disease entities which may be considered in the majority of cases to be specifically initiated by the activity of the fusospirochetal organisms are necrotic gingivitis, Vincent's infection, postextraction infection, noma, balanitis, phagadenic ulcer, hospital gangrene, gingivitis, pyorrhea, chronic tonsillitis, tropical ulcers, pulmonary abscess, pulmonary gangrene and Ludwig's angina.

The gingival sulci are the areas primarily invaded by these organisms, and the site of their continued existence in the oral cavity. Once infected, they constitute permanent foci which may initiate at any time, in addition to the underlying destruction, an acute local reaction or allow for the development of a remote fusospirochetal localization. In view of the extreme importance of the initial gingivitis, the classification in Table 2 is offered.

Chronic specific gingivitis is an important condition *per se,* but potentially it has almost unlimited possibilities for the production of disease. The treatment and prevention of the spirochetoses is a very important and almost exclusively dental problem, for it is by the proper treatment of the initial gingivitis that remote fusospirochetal localization, pyorrhea, the development of nonspecific periodontal foci of infection and their various sequelae can be prevented from developing.

The spirochetoses constitute one of the least recognized and most unsatisfactorily treated of the disease entities. The concept of the chronic forms of this disease is so new and the symptomatology

TABLE 1.—CLASSIFICATION OF SPIROCHETOSES

Gingivitis	95 per cent specific	(A)	Vincent's stomatitis Scorbutic stomatitis Mercurial stomatitis Bismuthosis (gingival)	Chronic
		(B)	Subacute Moderate	Chronic
		(C)	Mild Chronic Latent Endemic Unrecognized Incipient Gingivitis of pregnancy	Chronic
	5 per cent nonspecific	(D)	Chemical Thermal Mechanical Systemic (specific organisms) Bacterial (generally associated) Anemic	Chronic

and pathology so vague and indefinite that it is readily understandable why, in the absence of an acute reaction, an epidemic or microscopic studies, it is so frequently overlooked.

Hirschfeld[12] well sums up the salient points in the diagnosis of chronic active gingivitis: "The characteristic clinical symptoms which lead me to consider a condition as probable chronic Vincent's

TABLE 2.—CLASSIFICATION OF GINGIVITIDES

Spirochetoses	Superficial, accessible, localized	Acute	Necrotic gingivitis
			Vincent's infection
			Postextraction infection
			Noma (early)
			Balanitis
			Phagadenic ulcer
			Hospital gangrene (early)
		Chronic	Chronic gingivitis
			Pyorrhea
			Tonsillitis (specific)
			Tropical ulcers
	Remote, inaccessible, penetrating	Acute	Pulmonary gangrene
			Pulmonary abscess
			Sinusitis
			Meningitis
			Ludwig's angina
			Noma (late)
			Hospital gangrene (late)
		Chronic	Bronchiectasis
			Otitis media
			Empyema
			Appendicitis
			Osteomyelitis

For the proper control of the spirochetoses, one of the chief problems is that of educating the dental and medical fraternities to the recognition and significance of the chronic forms of this disease.

infection are a generalized chronic interproximal gingivitis with easily produced hemorrhage, and a history of intermit-

12. Hirschfeld, Isador: D. Items Int., 54: 731 (Oct.) 1932.

tent tenderness and increase of hemorrhage."

The significance of chronic, specific gingivitis is well expressed by Reasoner and Gill[13]: "Vincent's spirochete may be the inhabitant of the apparently normal buccal cavity. Its presence under these conditions is a potential source of danger to the person harboring it and to others closely associated with the carrier."

The present unsatisfactory status of the therapy of the fusospirocheial diseases is attested by the enormous number of treatments that have been and are being developed in the attempt to control it. Vincent's infection has by itself called forth a more diversified therapeutic armamentarium than all the other infections of the buccopharyngeal cavity put together. As a result of this chaotic condition, the spirochetoses are all together too frequently being cared for by the exodontist, surgeon and mortician.

From the nature of this infection, initiated primarily as a gingivitis and secondarily capable of causing remote localization almost anywhere in or on the body, we can appreciate the necessity for close cooperation between the medical and dental fraternities, if this disease is to be properly controlled. The steadily increasing incidence of this infection, the morbidity, and mortality primarily and secondarily following from specific and nonspecific periodontal disease, demand this cooperation. The necessity for this cooperation is well stated by Albray[14]:

> There has been considerable discussion relative to a closer cooperation between physicians and dentists. The problems of focal infection have interested both professions, and results obtained have demonstrated the necessity for and the value of such cooperation.
>
> A consideration of Vincent's infection shows that the physician and dentist, by working together, can render far more effective service than could otherwise be accomplished. As this infection usually starts in the oral cavity, about the teeth, its treatment there should be undertaken by the dentist. However, the dentist who wishes to render to his patients intelligent and conscientious service, should call on the physician to administer systemic medication, and by so doing, assist in the cure and prevent the spread of the infection. Furthermore, as Vincent's infection often complicates or accompanies systemic disturbances, consultation with a physician will in such instances prove of distinct advantage. Conversely, the physician who recognizes Vincent's infection in the mouth of a patient should feel it to be his duty to ask the dentist to assist him. The necessity for this is obvious, for conditions of a strictly dental nature with which the physician is not familiar, have a marked bearing upon the lesions of this infection, and no amount of treatment will avail in effecting a cure until these have been corrected.

In general, the spirochetoses may be divided into two groups, the local or the accessible, and the remote or inaccessible. The local and accessible group may be divided into two classes: the superficial and the penetrating. This division is purely schematic, for there can be no absolute separation of the spirochetoses, since a local lesion may at any time become penetrating and inaccessible.

We may state with authority that, for remote fusospirochetal localization, there is no other treatment than the parenteral injection of various combinations of the arsenobenzenes and the salts of bismuth, followed by whatever symptomatic treatment may be necessary. For the treatment of the accessible spirochetoses, two types of treatment are available, topical and general. Ordinarily, accessible spirochetoses should be treated topically. In any lesion which is or ap-

13. Reasoner, M. A., and Gill, W. D.: J. A. M. A., 88:716 (March) 1927.

14. Albray, R. A.: Dental Cosmos, 73: 878 (Sept.) 1931.

pears to be penetrating or may penetrate, the systemic injection of the known spirocheticidal drugs is immediately indicated.

Farber[15] states:

> While doubtless the condition in a majority of cases will sooner or later yield to local chemical treatment and in some cases to simple thorough cleansing, in a considerable number it almost interminably resists such therapy, nor is it an easy matter in the individual case to state in advance whether or not local treatment is going to be effective. It is well to remember that, in a certain percentage of cases, the disease advances more or less rapidly to serious stages, and may even threaten life.

Since this paper deals particularly with the treatment of the oral spirochetoses, which are generally superficial and accessible, and for which local treatment is generally sufficient, topical medications will be discussed in detail.

For the successful topical treatment of any bacterial infection, it is necessary, as stated by Kolmer,[16] that the chemical reagents used exhibit to a certain degree the following properties:

> They must have some degree of rapid bactericidal and bacteriostatic power in vitro; they should have some ability to destroy toxins; they should not be neutralized by body fluids or tissues; they should have the power to penetrate the cells of the body and to be free, in therapeutic amounts, of local or general organotropic—toxic—effects; they should attract phagocytes, leucocytes and monocytes to the site of the lesion and stimulate the surrounding tissue to proliferative activity.

In the treatment of a particular infectious process for which specific chemical reagents are available, it is possible to demand that additional properties be exhibited.

15. Farber, H. K.: Am. J. Dis. Child., 36: 463 (Sept.) 1928.

16. Kolmer, J. A., in Jordan and Falk: Newer Knowledge of Bacteriology and Immunology, p. 1101. Chemotheraphy with Special Reference to Treatment of Syphilis, page 361-364. Arch. Derm. & Syph., 21: 394 (March) 1930.

For the etiologic treatment of the oral spirochetoses, the chemical agents used must be evaluated by their demonstration of the following properties: specificity, nontoxicity (in therapeutic amounts), penetration, prolonged effect, dissolving action on necrotic material, rapidity of effect, length of time to effect a cure, analgesic effect, freedom from caustic or destructive tendencies, ease of application, cosmetic and esthetic value (not unduly unpleasant to patient), cost and prophylactic value. Lastly, and of the utmost importance, it must be correspondingly as efficacious in the treatment of the severe forms of the disease as it is in the mild cases.

For its evaluation, a reagent which is used in the treatment of any disease for which a specific exists must be submitted to examination according to the standards by which the latter is judged.

As a prelude to our consideration of the treatment of the oral spirochetoses, we wish to state that, in the vast majority of cases, the onset is insidious. If the onset is acute or if a low-grade infection is activated, the tendency is for them to pass into a symptomless chronic state without treatment.

Smith,[5] in speaking of fusospirochetal angina, states:

> Most cases, if left untreated, eventually recover. The mild diphtheroid type usually recovers in from two to six days. The more severe ulcerative type persists for one, two or even three weeks if left untreated. The chief reason for making an early diagnosis and treating these lesions promptly is to avoid the development of complications, which may be very severe and which not infrequently are fatal.

Considering that the disease is generally selflimited, as far as the acute symptoms are concerned, it is easy to understand why so many different methods of treatment have been recommended. It would appear both hasty and unwise

to recommend to the profession a new treatment for the spirochetoses until it had had wide and successful clinical application, particularly in the treatment of the more severe forms of this disease.

From a review of many of the "cures" offered, it is apparent that the condition ofttimes improves in spite of treatment.

There are four groups of medicaments which have achieved quite general use in the topical treatment of the localized accessible spirochetoses (ultraviolet therapy will not be mentioned other than to state that we consider it to be without merit in the treatment of the chronic forms of this disease, and definitely contraindicated in the acute forms): 1. The general antiseptics: metaphen, mercurochrome, iodine. 2. The cauterants: chromic acid, acetic acid, silver nitrate. 3. The oxydizing agents: sodium perborate, hydrogen peroxide. 4. The spirocheticides: arsenobenzol, Fowler's solution (solution of potassium arsenite), bismuth salts.

In any accessible infectious process, the topical application of the common antiseptics should, generally speaking, be expected to be beneficial to a certain degree. The spirochetoses do not constitute an exception, and the mild forms may be controlled and apparently cured by these agents. The lack of specificity of the general antiseptics, their inability to effect a rapid cure; the frequency of severe complications and the persistence of chronic infections following their use, and their valuelessness in the acute forms of the disease do not make them an acceptable means of treatment.

In the chronic forms of this disease, which are secondary infections, the use of general antiseptics is indicated as an adjuvant to spirocheticidal therapy. Metaphen has proved satisfactory in these cases, rapidly promoting the eradication of secondary infectious agents associated with the chronic oral spirochetoses.

Thompson,[17] using a modified Keilty technic, has recently demonstrated the relative values of the antiseptics used in the treatment of gingivitis. Cultures from the gingivae were inoculated into mediums containing the various antiseptics.

Sixteen drugs were used in the tests, with the following results (100 per cent indicates complete inhibition of growth): metaphen, 100 per cent; merthiolate, 100; acriviolet, 100; gentian violet, 100; acriflavin, 97; subidin, 58; chromic acid, 53; rivinol, 31; argyrol, 21; hexylresorcinol, 16; detoxol, 6; mercurochrome, 0; neosilvol, 0; copper sulphate, 0; potassium permanganate, 0; sodium perborate, 0; controls, 0.

From these data, the clinical response following the use of the various antiseptics can be satisfactorily anticipated.

CAUTERANTS

The cauterants have little to recommend them in the treatment of the spirochetoses. Any good result which follows their use is derived from the nonspecific destruction of the tissues and the organisms contained therein. Many times, it is not only impracticable, but also mechanically impossible to sterilize a focus of infection by their use. Their action is momentary and limited only to the area actually cauterized. The organisms outside that immediate field are not detrimentally influenced. On the contrary, the tissue destruction promotes anaerobiosis, which reacts to the advantage of the fusospirochetes and makes complications more frequent and serious. Because their use makes later and better advised treatment more difficult, we feel

17. Thompson, W. E.: D. Cosmos, 74: 1090-1091 (Nov.) 1932.

that the cauterants should be limited to the treatment of localized, minute, superficial lesions which can be completely sterilized at one sitting. A 10 per cent solution of chromic acid is quite efficient.

OXIDIZING AND TEMPORIZING AGENTS

Within the last few years, the oxidizing agents, used alone or in conjunction with the arsenicals, have attained widespread use in the dental profession for the treatment of Vincent's infection and stomatitis. Of the various agents in use for the treatment of the oral spirochetoses in the United States, sodium perborate has gained the widest acceptance and is today perhaps more generally used for the treatment of this condition than any other drug.

Sodium perborate was introduced into the practice of dentistry in 1913, by Henri Letard,[18] and has progressively increased in favor in both the medical and the dental profession. An idea of the present status of sodium perborate therapy may be gained by quoting from Beckman[19]:

> Bloodgood, whose mouth clinic at the Johns Hopkins Hospital has become quite well known, states that this drug will rapidly cure more than 95 per cent of cases if properly applied. He writes: "I would not have any difficulty in presenting at my weekly clinic one or more examples of sore mouth in which the cover slip examination revealed the organisms of this disease predominating, and I would be able to present the same patients almost completely well at the next clinic, or within two weeks." One is inclined to be just a little skeptical of such enthusiasm and to wonder what proportion of these patients would still be "cured" after the expiration of another interclinic period and still another. However, since the Vincent organism is anaerobic, the use of sodium perborate, which liberates large amounts of oxygen, is rational; indeed, in the hands of all who have used it faithfully, it seems to have given better results than anything else.

For the proper consideration of the treatment of spirochetoses, it is essential that the underlying pathologic process be understood.[3]

Generally speaking, a cross-section through any fusospirochetal process demonstrates three recognizable zones. First, there is superficial necrotic layer composed of dead cells and filled with innumerable bacteria of all varieties. Fusiform bacilli are infrequent and spirochetes rarely found. Immediately below this is the seminecrotic layer in which cellular differentation is still possible. The flora is mixed, cocci, vibrios and other nonspecific organisms predominating. The fusiform bacilli are plentiful and the spirochetes are not rare. In the deep layer, the fusospirochetes are the only microbes present and the spirochetes can be seen leading the fusiform bacilli in the invasion of the normal tissue.

Most anaerobic organisms, on exposure to air, utilize oxygen for the formation of hydrogen peroxide and, by so doing, spontaneously limit their own growth. From this consideration, it would appear that the topical application of the oxygen liberating materials would exert a beneficial reaction on these infections by causing a relative specific destruction of the anaerobic organisms. This is true to a degree, and nascent oxygen does influence these organisms deleteriously, but, unfortunately, most anaerobic bacteria, the fusospirochetes included, can withstand aerobic conditions for a time. Again unfortunately, the fusospirochetes lead in the microbial invasion and, by the time there is a symptomatic demand for treatment, it has insinuated itself deeply into the tissues and can neither be reached nor be influenced

18. Letard, H.: D. Digest, 15: 478, 1914.

19. Beckman: Treatment in General Practice, p. 267.

by topical applications of the oxidizing agents. The outcome of the infectious process will depend on the virulence of that particular strain of organisms and the resistance which it encounters.

Considerable clinical experience with the nonspecific reagents, and sodium perborate in particular, makes us more than skeptical in the face of the enthusiasm of the various workers who are using these materials. We found that, even after long and intensive use of these reagents, we could not cause the disappearance of the causitive organisms from the oral cavity. Similar observations are reported by Stafne, as quoted by Musburger[20]:

> One thing I haven't been able to do is to get rid of the organisms at any one time, not even for a day. Some of the men who are making a study of it claim that they stop treatment only when they get a negative smear on three successive days. I don't know what methods they use in preparing these smears, but I use a double check, using a dark-field and several prepared smears (stained) at each sitting, and if I didn't find them in one, I did in the other.

Sodium perborate alone does not sufficiently shorten the course of the disease. The reported cures of 95 per cent of these infections in from one to two weeks by the use of this drug does not compare overly favorable with the results secured by Nature unassisted, who, in a similar period of time, secures almost identical results.

It has been our experience that the reagents liberating the greatest amounts of oxygen were the most satisfactory, particularly chromic acid, which liberates 24 per cent, and potassium permanganate, which liberates 16 per cent. We have ascribed the good results, when secured, to the cauterizing effect on the tissues and the organism, rather than to a specific destruction of the microbes by the oxygen.

As demonstrated by Thompson,[17] the only good effect which could possibly result from the use of sodium perborate would be the changes brought about by the liberated oxygen. It is not particularly active in this capacity, freeing less than 10 per cent. Indeed, as Hardgrove[21] states: "It is not active enough to be really considered a specific, or to bring about a sudden termination of the disease."

The interplay of several factors prevents the oxidizing agents from becoming an acceptable means of treatment for the oral spirochetoses. The topical application of these reagents is generally irritating, frequently painful and, if concentrated, causes tissue destruction. Their action is momentary; they do not penetrate; they have no dissolving action on the necrotic material; they allow for the development of chronic infection; complications are not prevented; healing is not rapidly promoted; and, finally, they are valueless in the more severe forms of the disease. The oxidizing agents do not meet to a sufficient degree the nonspecific properties listed by Kolmer[16] nor the specific requisites listed by us, to make them theoretically ideal reagents for the treatment of this disease. Our clinical experience with these drugs demonstrates the validity of that conjecture. The danger from emboli, following tissue destruction by the rapid liberation of oxygen in a spirochetal pocket, contraindicates their use in closed cavities. Further, they are sharply limited in their application. Even the most sanguine observer cannot admit of their having value in the treatment of the re-

20. Stafne, quoted by Musburger, L. E.: D. Cosmos, 70: 520-527 (May) 1928.

21. Hardgrove, T. A.: Vincent's Infection from Pathologic and Bacteriologic Point of View, J. A. D. A., 15:203 (Nov.) 1928.

mote, inaccessible and acute penetrating "noma-like" spirochetoses.

Some writers recommend that they be used in conjunction with the specifics. We do not believe that healing is more rapid when they are so used, and see no necessity for thus complicating therapeutic procedure.

If any oxidizing agent is to be used, we favor chromic acid, the cauterizing effect of which does, in a majority of cases, have a beneficial influence on the course of the disease. In view of the disadvantages of this method, and the more satisfactory results secured from other forms of treatment, we cannot recommend it.

SPIROCHETICIDAL DRUGS

In consideration of the common fusospirochetal etiology of the spirochetoses and the specific destructive action of the arsenicals and salts of bismuth on the fusiform bacilli and the spirochetes, the main organisms in this symbiotic process, it is but natural to assume that these reagents should be of value in the treatment of the infections caused by this group of bacteria.

These drugs have, in our hands, and in the hands of many others, demonstrated their value, and today they are universally recommended and considered as the best means of treatment for the spirochetoses.

Our results over a period of years in the treatment of the various manifestations of this disease entity have been such that we may state that suitable combinations of the arsenicals and salts of bismuth will promote the rapid healing of all but the acute penetrating "noma-like" forms of this disease. In the penetrating cases, they afford the only possibility of medical cure. If they cannot be controlled by the specifics, we may be sure that they will not be favorably influenced by any other drug.

Lack of space limits a consideration of the other recommended remedies. In order that their relative status may be understood, we insert the following quotations:

Beckman[19]:

Miscellaneous remedies: practically all the antiseptics and disinfectants have at one time or another been advocated for local use in this disease. Those which have had the greatest vogue are tincture of iodine, 10 per cent chromic acid, 10 per cent silver nitrate, 1 per cent potassium permanganate and 2 per cent methylene blue. The results obtained with none of these substances warrant special mention here.

Kolmer[16]:

Various local applications have been advocated from time to time for the treatment of this infection, among which may be mentioned applications of solutions of copper sulphate, potassium chlorate, iodine, chromic acid (2 per cent), etc., also powdered methylene blue, Dakin's solution, acriflavin, etc. Arsphenamine and neoarsphenamine, however, have yielded particularly good results and especially by local application.

The etiology and treatment of these conditions have been satisfactorily understood in Europe and South America for some time, but it has been only recently that American investigators have given much thought to the problem.

The arsenicals were first introduced into the treatment of oral disease by the Chinese 2,000 years B.C., but it was not until after Ehrlich,[22] in 1907, demonstrated the specific destructive action of the arsenobenzenes on the spirochetes that they came into general use.

The intravenous injection of arsphenamine in the treatment of Vincent's infection was introduced in 1910 by Ehrlich.[22] In the following year, Achard and Flandrin[23] began the topical use of

22. Ehrlich, P.: München. med. Wchnschr., 57: 2268, 1910.

23. Achard and Flandrin: Bull. et mem. d. hôp. de Paris, 31: 504, 1911.

this material. At about the same time, Rolleston[24] introduced the glycerinated solution of arsphenamine for the treatment of angina and "trench mouth," which became the standard method during the war.

Albray,[14] who has had considerable experience with the arsenobenzenes in the treatment of Vincent's infection, wonders why this form of treatment is not more generally used by the dental profession.

> For some unexplained reason the use of the arsenicals in the therapy of Vincent's infection has not been generally accepted, although there is plenty of evidence as to the efficacy of these preparations. I have never had anything occur as a result of using them to indicate that they were in any way harmful to the patient. Quite the reverse, for by their use, this distressing condition is cleared up in much less time, with less permanent damage to the structures involved than by any other means; also, the pain and discomfort is usually relieved within an hour or two after the first application of arsphenamine to the lesions.
>
> There is justification for internal therapy, because the organisms work their way into the tissues, where they are most active in causing destruction. Application of the drugs to the surface alone cannot reach them.

Since their introduction by Ehrlich,[22] the use of these materials has spread rapidly and, at the present time, except in gingivitis, they are accepted as the medicament of choice in the treatment of the spirochetoses and are recommended by almost all authorities.

The chief problem confronting the dentist and the internist in the treatment of these diseases is their recognition. Once the unity of the spirochetoses is accepted and their significance understood, there are any number of drugs which will specifically control the disease. Kolmer[16] states: "For the treatment of fuso-spirochetal ulceromenbranous angina, these compounds (arsenobenzols) may be regarded as specifics insofar, at least, as concerns the part played by the spirochetes." Mangabeira - Albernaz[4] states: "Whenever and wherever the fuso-spirochetic combination is causing a pathological process, ear, ulcer or mouth, the treatment should be specific, that is, the arsenicals and bismuth."

Voss[25] is of the opinion that "the chief drugs to be remembered are the spirocheticides, which include potassium arsenite, the arsphenamine group and the salts of bismuth."

Strong[26] expresses himself similarly: "No preparation other than salversan, neoarsphenamine, or arsphenamine has been shown to be more satisfactory for the treatment of a number of spirochetal diseases, though a number of other organic preparations of arsenic, antimony, or bismuth have been employed in treating some of the affections with almost equally good results."

Kritchevesky and Seguin[1] state:

> The unity of the spirochetoses is further evidenced by the efficacy in all such diseases of the antispirochetic medicaments, more especially the arsenobenzols. Since Ehrlich first successfully treated a case of Vincent's angina by arsenobenzol, these medicaments have entered the field of the regular therapeutics of acute oral spirochetoses of the mouth and, with the exception of noma, regularly produce the most satisfactory results.

Kolmer[16] recently reported that bismarsen has yielded encouraging results in the treatment of the more severe cases of Vincent's angina.

24. Rolleston, J. D.: Proc. Roy. Soc. Med., 1913, p. 711.

25. Voss, Harrell: Arch. Otolaryngol., 14: 1-8 (July) 1931.

26. Strong, R. P.: United Fruit Co. M. Dept. J.

ORAL DIAGNOSIS AS IT FUNCTIONS TODAY*

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ONLY through a proper understanding of the field of oral diagnosis can we visualize its merits and deficiencies as practiced at the present time. Let us divide dentistry into two sections: (1) therapeutics, dealing with operative dentistry, crown and bridge practice, prosthetics, oral surgery, periodontia, orthodontia, etc., and (2) oral diagnosis. It should be borne in mind that these are not isolated divisions. They overlap, and a thorough understanding of one is not possible without a working knowledge of the other. All therapeutic measures are based on oral diagnosis, and oral diagnosis is an integral part of every phase of dentistry. Again, oral diagnosis must use as its tool the basic sciences, such as histology, pathology and bacteriology, and through a clear perception of the facts of these sciences in coordination with what constitutes proper procedure in practice, we may hope to arrive at intelligent diagnosis.

Moreover, oral diagnosis considers the mouth as a unit in relation to the rest of the body. Mouth health and systemic health are dependent on each other. Therefore, our first consideration in making a diagnosis must be the elimination from the mouth of all pathologic processes which might have a bearing on systemic disease. Also (and this is of utmost importance), there enters here the idea of prevention of systemic disease as well as of oral disease. Let us call this the tearing-down process. Next, there is the task of restoring the mouth as a masticatory unit so that it can maintain itself in a state of health and cleanliness. This may be called the building-up process. During the tearing-down process, we must keep in mind the building-up process, for there are certain patterns of mechanical devices which function better than others. For example, some teeth may be sound units in themselves, but it is wise to eliminate them if they serve no useful purpose, or, in fact, act as a hindrance to the construction of a well-balanced, well-designed mechanical appliance.

Thus, it is essential in tearing down to so leave the mouth that a favorable reconstruction may be made. This brings into play a knowledge of occlusion, of dental anatomy, of periodontia, of orthodontia, etc.

Now how is this plan put into effect today? The average practitioner sets out to make a diagnosis. First of all, he may or may not require a set of roentgenograms. If he has demanded them, he proceeds to interpret them to the best of his ability, which very often is wofully limited. The prevalence of such pseudo-efficiency may be assigned largely to the lack of facilities for teaching roentgen-ray interpretation in some of our institutions, or to the fact that many dentists acquiring roentgen-ray equipment are satisfied with the instructions received at the hands of the concern selling them the outfit.

*Read at the organization meeting of the American Association for the Advancement of Oral Diagnosis, Buffalo, N. Y., Sept. 12, 1932.

The latter idea also explains the misuse of other aids in diagnosis, such as transillumination. Instead of using these devices for the purpose for which they are intended, they are generally applied as short-cuts.

After the roentgen-ray examination, teeth are extracted and restorations undertaken. The routine here becomes a business transaction. So many spaces to be filled, so many holes to be plugged at so much per hole. The dental diagnosis takes on the form of a bill for goods to be delivered. It does not concern itself with occlusion or any of the other factors before mentioned.

Remaining teeth are used as units on which to attach replacements without regard for the ultimate health of abutment teeth. Teeth are retained and treated when their immediate extraction would preserve the alveolar ridge for future dentures. In addition, the practice of pulp testing is not generally understood and not sufficiently used. This aid in diagnosis should be emphasized and developed.

A credit report takes the place of what should be the case history. The charting of the mouth consists merely of the financial record. Even the conscientious practitioner who wishes to preserve necessary data on his cases is at a loss as we have no uniform system of charting. To the man in California, a chart prepared by a man in New York is of little value.

In the eyes of his patient, the diagnostician occupies a humble place. His position lacks the dignity of importance. In most instances, there is no charge for his advice. Patients have been taught to receive bids on the repair of their mouths just as they ask for bids when building a garage. They go from dentist to dentist shopping for a price. Usually, the favors are granted to the lowest bidder.

Is this attitude the same when an individual consults a physician? Certainly not. The medical man's diagnostic ability is precious and is so recognized. The fee for his opinion is a foregone conclusion.

In recent years, many departments of dentistry have made rapid advancement but oral diagnosis has not kept pace. Oral diagnosis, like diagnosis of systemic diseases, should be on a par with every other phase of dental practice. Yet, in many dental schools, it is not even listed in the curriculum; while, in others, it is regarded merely as a means of routing patients. The important consideration of what is to be done to a mouth to preserve its integrity and safeguard its future health, as well as the health of the individual, is often without name or category.

This, in short, is a sketch of the manner in which oral diagnosis functions today. Remedial measures are obvious. And as an aid toward learning and education in this field, the Association for the Advancement of Oral Diagnosis should be a constructive step.

PRACTICE MANAGEMENT AN OFFICE ASSET*

By JOHN F. CHRISTIANSEN, M.S., D.D.S., Los Angeles, Calif.

EVERY successful practice acquires a certain distinctive personality. It is a reflection of the personality of the dentist who utilized his natural intuitive and trained ability as an approach —it is a reflection of an energetic working or moving cause. Professional success is based upon constant endeavor to improve, to give fair treatment and to render a public service of the highest order.

To enumerate all essentials for professional success in dentistry, or in any other vocation, answers no good purpose. There are probably scores of opinions relative to the meaning of success. Personal satisfaction found in the work in which one is engaged, the mastering of technical procedures, the sense of work well done, efficiency in management may be considered as some of the marks of success, each by itself or all in combination.

Interest or personal satisfaction in professional life has an almost inexhaustible range. Financial income or what it will bring does not devastate the field of interests. To reduce interest in professional activity to "profit motives" only, and thereby fail to evaluate the "service-motives" mentioned, would destroy most of the happiness found in our work and, additionally, it would probably deter professional progression. Admittedly, the distinction between the two motives, profit and service, cannot always be sharply drawn, even though it is well understood that financial gain should be a subordinate consideration. These two motives are not conflicting, but supplementary. Both may be called incentives. Obviously, there should be opportunity for both professional reward and prospective financial gain to compensate those in whom distinctive trust and on whom obligations are placed.

Without worthy incentives, efficient effort will probably diminish. Supplementary to each other as service and profit are, it is generally through the latter that misunderstandings frequently arise both outside and within the office. A terse definition of profit is given, and the word will be thus used within the following paragraphs. "Profit in theory is the compensation which society makes to an industry for rendering service, as distinguished from the direct cost of the service." Profit is an indispensable element in a business system, and the same with certain modifications applies to professional practice. Likewise, a business that produces no profit is no business.

If it be held that there is too great a disparity between the opportunities for financial gain and professional reward in office affairs, there should be within that office a greater concerted effort in office organization toward equalizing these

*Read before the Section on Economics and Office Management at the Third Triennial Session of the Pacific Coast Dental Conference, Seattle, Wash., July 8, 1932.

factors. Control from within, if it can be secured, is always safer, wiser, more thorough. Control from without should, of course, never be condoned, except perhaps where strictly social conditions are primarily or absolutely responsible. A profession, such as ours, that is legally protected from competition, and, therefore, more or less amenable to written law, whose public relationships are conspicuously censored by its own organization councils, has ever been conscious of its duties and its obligations to render adequate service. For the financial welfare of the average practice, more practical training in personal professional business management is a necessity. This training should be properly sponsored and acceptable to our organizations. It should be initiated and emphasized in the schools, beginning in the first year of college work and continuing through progressively and intensively to the end of the graduate course.

Some have gone so far as to assert that professional service should be paramount and that systematic and efficient office management is of trivial importance. Public opinion is of importance, for in actuality we are the servants of the public and more or less subject to its control. It is the constant contact with the public that engenders and stimulates the study of office administration, especially in the average general dental practice. The objective should be to systematize office management so that waste of effort and time is avoided, and thus provide in part, perhaps, against errors in judgment, particularly relative to the disparity between the services rendered and the financial returns.

The professions, not unlike other forms of human endeavor or industries, have their own peculiar problems relative to production, disposal and placement of services. All modern enterprises have a general background of economic and sociologic relationships. It is toward these additional responsibilities, outside the strictly professional attainments, that education might be more generously directed.

The suggestion is not offered with the thought of merging schools of commerce or business administration with professional or vocational institutions. It is intended merely to emphasize an academic business training to decrease the handicaps of having to acquire empirically the knowledge and experience essential to the management of an office practice. Conversely, I am bearing in mind the futility of attempting to train dentists to become business experts or accountants of the plain or certified varieties.

The idea of the academic training course in personal professional management is to help the dentist to avoid the perplexities met especially in the initial years of practice. While to give useful information for this purpose alone is a responsibility of the schools, it is not exactly a parental responsibility.

During the past ten years, much effort has been made to work out a science of personal professional management. Training is one thing; education is another. In itself, rigorous training in professional management will probably give a maturity of judgment. At least, a development of better and keener judgment will be hastened. It will present a means through which certain practice building factors may be sensed quickly. It probably will tend to curtail invisible economic weaknesses in office routine administration. It may furnish reliance for plans for the current period and the future; and, of principal importance, it will dictate a method for

imparting a technic of sound appraisal of office work together with a decidedly better knowledge of business standards, and, too, it will give the real individual office reasons for them. This training may be advantageous as an "underpinning" in business relationships and a guard against any necessity for pecuniary trickery. Unless this information is distinctly impressive, the idea that all professional financial success is dependent on the survival of the shrewdest will still linger. Training in office management has significant value, and, last but not least, it develops professional obedience to established rules and produces a more correct office control. Financial remuneration is not to be accredited entirely to technical dexterity or scientific accomplishment. Studious training in executive management should be given its portion of credit for the comparative success of a practice.

Professional enthusiasm should not lead to a disregard of requirements in the field that this subject aims to teach. No doubt the mental reward from professional devotion is more lasting than any other form of recompense, but this mental reward will eventually dwindle and discouragement result if business elements are not constantly borne in mind.

As before mentioned, in dentistry or in any other profession, success is relative. Individual satisfaction and happiness in the work may spell success to one dentist and not to another. Even when skill and personality are comparable, financial returns often show dissimilar results. Certainly no office practice can be considered a success on the basis of production results alone. Net financial returns represent frequently a difference in management, not a dissimilarity in professional principles or in technical and scientific attainments. As an illustration of differences in management the accompanying comparative office statements are presented.

PROFIT AND LOSS STATEMENT
COMPARATIVE REPORT
CASE OF DR. A AND DR. B

	"A"		"B"
Gross Earnings:	$978.00		$813.00
Laboratory and Supplies	$165.00	$75.00	
Rent	145.00	67.00	
Wages	200.00	20.00	
Telephone	21.00	9.00	
Laundry	11.00	6.00	
Business Office	18.00	15.00	
Depreciation	91.00	651.00	33.00	225.00
Net Profit	327.00		588.00
Hours Productive	142		82
Hourly Fee	6.83		9.91
Hourly Cost	4.58		2.74
Hourly Profit	2.25		7.17
% Expense to Gross	66		27

"A" has his own suite of offices (two operating chairs, private business office, separate sterilization rooms and expensive office furniture). "B" is associated with another dentist and a physician, each of whom pays portionately for rent, telephone operator, telephone charges, etc. "A" employs two assistants: a girl who has charge of his business records and reception room, another who is his private assistant at the chair. "B" has no assistant. "A" has twenty scheduled chair appointments a day.

PROFIT AND LOSS STATEMENT

No._____ Month of_____

GROSS EARNINGS:		
Dental	610.75	
X-ray	.....	
Miscellaneous		610.75
Less Professional Discount		.....
Total Gross Earnings		610.75
PRODUCTION EXPENSES:		
Laboratory (Commercial)	71.24	
X-ray	.....	
Supplies	11.97	
Precious Metals	19.96	103.17
		507.58
MISCELLANEOUS AND OPERATING EXPENSES:		
Laundry	8.38	
Telephone	8.72	
Office Salaries	78.00	
Light and Power	.....	
Rent	98.50	
Taxes	.....	
Insurance	.....	
Business Office	5.00	
Auditing - Legal	.....	
Interest	.....	
Miscellaneous	6.08	
.....	.....	
.....	.....	
.....	.....	204.68
		302.90
Depreciation	67.17	
Interest on Investment	.....	67.17
TOTAL NET EARNINGS		234.73
Other Income:		
.....	.....	
.....	.....	
TOTAL INCOME		234.73

*Percentage of Expense to Gross Earnings 61%

Hours: chair 63:00	Gross Earnings 610.75	Hourly Fee 9.70
" laboratory		
Total 63:00		Hourly Cost 5.97
Hours not charged for	Total Expense 375.02	
Net Productive Hours 63:00		Hourly Profit or Loss.. 3.73

*To find Percentage add Total Production and Operating Expenses and divide by Total Gross Earnings.

There are a few who intuitively sense correct office management in all divisions of practice. Most of us lack this intuitive judgment and therefore the study of economics is important. The office management phase of a practice aims at household efficiency. Office management as here given includes all factors employed in the production and completion of services not connected with actual professional skill or ability.

It may be interpreted that the thoughts expressed are directed toward the idea of placing the dentist in monetary splendor. We are not discussing the problem of fees. It is rather intended to emphasize the value of proper utilization of time to meet the demand for services and also to so systematize the internal administration of the office that the efficiency of professional services will not suffer, and yet the financial relationships will not be disregarded. The system which we suggest, or any other, is not a royal road to success, but mostly a means of emphasizing that these divisions (professional and financial) always exist in a practice and that both should be recognized without disturbing an economic balance. Sociologic conditions are responsible for the demand for services. Rules and principles for conducting a practice are based upon this theory.

Even with the acknowledgment of meritorious accomplishment in office business administration, there is ample evidence that considerable work remains to be done in forwarding the idea of the necessity for the training in and study of this subject in most of our practices. Office practice analysis seems to be in its infancy.

Facts expressed by figures have telling animation provided there is acquired the ability to recognize their significance. Indirect help—"outside" accountancy—cannot displace the dentist's own thoughtful analysis and decisions based on the information revealed through his own individual office practice accounting. Properly interpreted, office records are particularly useful to the management in guidance and an inspiration to progressive accomplishment.

It is believed that the dentist himself should have a thorough understanding and a working knowledge of a system of accounting to be used as applied office economics. Each practice presents problems in management which in important features are not duplicated in any other office. These "feature-problems" must be solved on the basis of facts recorded in the written transactions of that particular office. As far as bookkeeping is concerned, the dentist should know (1) what is being done, (2) why it is being done, (3) how it is being done and (4) why it is being done that way.

The office records are the guides for the continuation of business or for the changes necessary to its continuation in an improved manner. Valuable as records are, they will not promote business. They are used for the purpose of limiting or facilitating function, for their daily working value, as indicated in the individual office. Office records are compiled in order that their effects (favorable or unfavorable) on the property and the rights of the business itself may be clearly displayed for consideration by the responsible management. Such information may be of considerable worth and useful to the betterment of the daily practice routine. Often, these aids determine for the dentist his own efficiency rating, and, likewise, the efficiency of office assistants as far as office production results are concerned.

The actual worth to the office of a system of bookkeeping is solely depend-

CONSOLIDATED RECORD OF COMPARATIVE FEES, SIX-MONTHS PERIOD

	Extraction			Amalgam			Inlay			Prophylaxis			Crown and Bridge			Silicate			Foil		
	No.	Min.	Amt.	No.	Min.	Amt.	No.	Min.	Amt.	No.	Min.	Amt.	No.	Min.	Amt.	No.	Min.	Amt.	No	Min.	Amt.
January	16	275	$58.00	17	655	$87.50	2	290	$30.00	17	640	$82.00	3	1165	$210.00	12	495	$76.00			
February	3	90	15.00	13	565	68.00	2	265	45.00	9	420	43.00	2	445	116.00	9	350	49.00			
March	6	155	28.00	19	665	80.00	3	430	50.00	8	275	36.00	3	850	218.00	5	220	25 00			
April	3	50	11.00	14	465	63.50	2	295	33.00	10	370	49.00	3	555	103.00	14	470	63.00	6	280	$42.50
May	3	65	13.00	13	530	65.50	5	555	78.00	11	445	55.00	2	640	350.00	12	430	58.50	9	750	85.00
June	5	120	17.00	14	545	62.00	5	450	77.00	13	490	61.00	3	720	131.00	7	285	32.00	10	560	65.00
Total	36	755	142.00	90	3425	426.50	19	2285	313.00	68	2640	326.00	16	4375	1128.00	59	2250	303.50	25	1590	192.50
Average	1	20	3.94	1	38	4.74	1	120	16.47	1	39	4.80	1	272	70.50	1	38	5.16	1	63	7.70
Fee per hour			11.82			7.50			8.23			7.56			15.45			8.10			7.32
Profit per hour			5.87			1.55			2.28			1.61			9.50			2.15			1.37

Total expense for period	$2,252.26	Total working hours	378
Production expense for period	619.13	Total working hours	378
Fixed expense for period	1,633.13	Total working hours	378

Hourly cost		$5.95
Hourly cost		1.63, A
Hourly cost		4.32, B

Production Expense

Laboratory	$427.45	
Precious Metal	119.86	
Supplies	71:82	$619.13

Fixed Expense

Laundry	$ 50.28	
Telephone	52.35	
Salaries	470.00	
Rent	591.00	
Miscellaneous	66.48	
Depreciation	403.02	1,633.13
Total Expense		2,252.26

The foregoing figures show:

A. Profit for amalgam fillings is greater than profit on inlays, if material cost is considered.

B. Profit on silicate fillings is greater than the profit on inlay restorations, if material cost is considered.

C. Profit for one hour on crown or bridge work equals profit derived from 1.7 hours extraction of teeth; or 4.2 amalgam filling, 3.4 inlay, 4.0 prophylaxis or 3.0 silicate filling.

D. The greatest ratio of profit in order is (1) crown and bridge work; (2) dentures; (3) extraction; (4) prophylaxis; (5) silicate filling; (6) amalgam filling; (7) foil filling; (8) inlay.

Gross income for period	$3,664.50	Average monthly income	$ 610.75
Average monthly expense	375.37	Average hourly cost	5.95
		Average hourly fee	$9.70

Percentages

Production expense to gross income	17
Fixed expense to gross income	44
Total expense to gross income	61

Office charge a productive hour, $9.70; net profit an hour, $3.70; office operation and maintenance cost including commercial laboratory expense, precious metals and production supplies, $6.00 a productive hour; average production hours a month, 63. If production hours are increased 50% or to 90 hours a month, production expense will increase 50%, and fixed expense percentage will decrease.

ent on the information presented by the records which can and will be used advantageously. If records are not put to work, they represent a constant expense for maintenance. Oversystematization in bookkeeping is thus possible.

On the contrary, just doing business all the time without developing facts as expressed by figures is a frequent source of business error. Accounting must not only tell whether money was made or lost, but also why. A mere record of cash received and cash paid out is certainly not sufficient. There are numerous office transactions that represent values other than the literal money received or money disbursed. These transactions are concerned with those of the office organization and the dealings between the office and the outside. Such transactions include things that affect profit directly, as depreciation, deferred payment, "charged-off" accounts and unnecessary cost and losses. All such accounts require special attention in recording. They are frequently overlooked or neglected. They may be termed invisible expenses because they are usually not noticed until serious pecuniary damage has been done. Such neglected accounting on this type of influence has a definite detrimental effect on profit of the business.

Some professional men use bookkeeping sparingly and with a primary idea of just satisfying the requirements of an income tax return. They are careless, in this sense. Their office records can be only partly or poorly understood. One such system is complete(?) with only the use of a big yearly calendar as a journal or day book and a bank checkbook. This person never tries to analyze his job. Bookkeeping is most important when it makes possible the analysis of our work. For example, one cannot correctly determine over a given period an equalization of fees that is just to the average type patients and to the management, unless the written records are used as basic information for judgment and decisions in the problems arising in routine practice.

Services may be dispensed below actual cost. Not even gratitude is expressed for such services because neither patient nor operator actually realized that the services were rendered below actual cost to the office.

No special division of fixed and production expenses is made in figuring the hourly cost of operations. This may place an additional burden on operations requiring no laboratory or precious metal expense, but nevertheless places the office accounting records on the safe side of "cost." It also allows a greater margin for additional cost not originally computed in the first estimates for the other operations in the list.

Production expense $619.13 plus fixed expense $1,633.13 is $2,250.26. Total expense divided by 378 working or productive hours is $5.95 operative cost an hour.

For example, for an inlay: nineteen cases completed for the six-months period, for a total fee of $313 gives a case average of $16.47. A total time for nineteen cases of 2 285 minutes gives an average of 120 minutes for each inlay.

The average inlay fee is $16.47, with an average operative cost an hour of $5.95. Two hours average time for an inlay yields $11.90, the average profit being $5.47, or $2.73 an hour.

It may be noted in the accompanying table that $403 depreciation for the six-months period is used. If the depreciation figure on the office equipment were totally disregarded, the example above would show the following result:

total expense including depreciation, $2,252.00, less depreciation $403.00 equals $1,849.00; 1,849 divided by 378 gives $4.89, hourly cost; inlay fee, $16.47; two hours at $4.89, $9.78 and profit, $6.69, or $3.34 an hour.

USES OF THE CONSOLIDATED FEE CHART

The consolidated fee chart (1) presents facts expressed by figures; facts which permit of comparison; tendencies and relationships; (2) gives the operator a chance to concentrate on operations on which the yield is in disproportion to certain other office operations; (3) affords a method of forecasting results, giving an opportunity for the development of dependable future policies (each office wherein such dependable figures are collected may overcome economic weaknesses in control toward a more uniform and profitable production volume); (4) presents an office "government chart" idea for the proper regulation of proportional fees, a critical analysis of results for the period stated; (5) presents definite references, a model for comparative fees for the various operations; (6) shows a "time-study" of efforts made under different conditions, a standard of performance for an individual operator, and (7) gives an estimate for future operation cost calculations.

PROFIT AND LOSS STATEMENT

As an aid to the office management, a profit and loss statement (1) affords an arithmetical method for supervision of office business; (2) presents data for arriving at a "business-judgment"; (3) gives a clear view of the consequences of financial acts and policies, a mirror of business transactions—a painting of the office management's efforts for a certain period; (4) shows what becomes of the gross earnings of a business for a stated period; (5) shows what salary the management should receive for the period in proportion to the volume of business shown by net profit thereon; (6) affords a comparison indicating whether business is going forward or backward, expenses being compared with previous periods of time; (7) shows the cost of gross earnings; (8) furnishes an expense standard or norm for the dentist's own particular circumstances (general statements of current practices of other dentists are not definite guides for an individual practice), and gives the history of the office in figures for the period covered.

Pertinent office information, proved accurate beyond question, is another principal reason that adequate bookkeeping should be maintained. A system should be replete in accounting for the entire office business trend; one that quickly and dependably portrays the results, favorable or otherwise, of the office business transactions.

The value of facts expressed in figures lies in the practical use that is made of them to guide the management in the production of profit volume. These figures are studied not for material value but for the functioning or daily working value as found in the dentist's office. A pertinent question is "Of what value is this record to me in the management of my business?" It is common sense to teach that what patients do not pay for, the dentist himself must pay for. Sharply speaking, this is where bookkeeping is very valuable, and dentists who have actually learned this truism through their own office accounting records have benefited thereby.

The lesson of a composite picture of office practices is that no detail of expenditure is too small to receive careful attention. The desire to render high quality service under the most pleasant

conditions, and to give almost no attention to business details, has constantly retarded the dentist in his financial income results.

In properly kept office records, one learns to know himself in office work and management. Such records are creative of individual self-reliance founded on a modern conception of justice to himself and to those whom he serves. Informative and dependable records are retrospective and prospective. It seems that they are the only means of experiencing the truth, even though it may be sought for its own sake. "Truth may set us free." It is written records that are defenders of business policies, defensive for the particular office management against the assaults of many, and an insurance against unjust criticism of a proportion of stated estimates for services to be rendered, or for justifiable dental fees.

When the possibility of accomplishment is understood, properly filled in records produce a stimulation toward better office management, and they are perforce a guidance and an inspiration in our work, all of which, in turn, will aid materially in the realization of a truly productive volume for the office practice.

THE ROLE OF VITAMINS IN THE TREATMENT OF CERVICAL HYPERSENSITIVENESS*

By C. J. K. RYAN, D.D.S., Ray Brook, N. Y.

HYPERSENSITIVENESS of the cervices of the teeth is attributed to mouth acidity, toothbrush abrasion, traumatic occlusion, metabolic disturbances or a combination of these conditions. Franken[1] advances the view that the condition is due to a deficiency in fat-soluble vitamins and calcium since the addition of these substances to the diet resulted in improvement. The present study apparently confirms Franken's observations and also emphasizes the value of vitamin C in the treatment of the disease.

Among the twenty patients with cervical hypersensitiveness included in this series, eleven were suffering from pulmonary tuberculosis in the early stage, five in the moderately advanced and two in the advanced stage of the disease, and, in the remaining two, the diagnosis was questionable. Cervical hypersensitiveness of one or more teeth was found in all instances, particularly to thermal changes and to sweets. The hypersensitive areas were detected by passing an explorer over the dried tooth surface. During the first month, the patients were given a liberal general diet. If the condition remained unchanged, they then received, in addi-

*From the New York State Hospital for the Treatment of Incipient Pulmonary Tuberculosis.

1. Franken, S. W. A.: Successful Treatment of Painful Erosion Cavities, D. Cosmos, 66:785 (July) 1924; Treatment of Sensitive Cervices of Teeth, J.A.D.A., 18:156 (Jan.) 1931.

CASE DATA

Patient	Sex	Age Years	Chest	Sensitive Teeth	Approximate Amount of Milk Before and After Treatment		Period of Treatment Months	Results	Remarks
R.M.	F	18	I	1	¼ pint	1 quart	1	Good	"White line" around the cervix of each tooth
S.M.	M	26	I	7	None	1¼ quarts	3	Condition progressed	Poor mouth hygiene
I.P.	F	17	I	2	1 pint for 2 years, none before	1 quart	2	No change	Patient readmitted recently; both teeth nonsensitive, drank approximately 2 quarts of milk daily, in intervening two years
J.R.	M	22	I	1	None	¾ quart	5	Good	
P.D.	M	21	II+	6	¾ pint for 8 months,none before.	1 quart	4	Good	
W.McK.	M	28	I	18	None	1½ quarts	5	Good	Yeast cake three times a day or two months
E.S.	M	22	I	2	None	1½ quarts	3½	Good	
E.N.	F	21	I	1	None	¾ quart	1	Good for one month	Tooth supporting partial denture with clasp
T.H.	F	25	I	1	None	1¼ quarts	6	Good for one month	Marked constipation
Ed.Ca.	F	21	Observation; cardiac	1	Very little	¾ pint	3	Improved	Very nervous
El.Cl.	F	35	I, pleurisy	10	None	¾ quart	4	Improved	
E.O'B.	F	20	III	12	None	¾ quart	6	10 good, 2 improved	Bone meal 40 grains a day, no apparent effect
T.C.	M	25	II	1	Little	1¼ quarts	7	Good	Tooth clasped by partial denture
M.D.	F	19	III	7	None	1½ quarts	8	Good	Intestinal tuberculosis
L.W.	F	26	II	1	None	1¼ quarts	8	Good	
M.O'K.	F	25	II+	2	Little for 3 years, none before	1½ quarts	9	1 good	Other tooth had large leaking alloy filling
V.N.	M	28	II	8	Little	1¼ quarts	6	4 good,others no change.	Poor oral hygiene
S.R.	M	27	I+	8	None	1¼ quarts average.	6	1 good,others no change.	Poor oral hygiene
O.P.	M	22	Observation	2	None	Very little	5	Good for 2 months.	Diabetes insipidus
					Without cod liver oil				
F.B.	M	20	I	2	None	1 quart	7	No change	

tion, thrice daily, one half ounce of cod liver oil and 3 ounces of tomato juice,[2] which contains not only the fat-soluble vitamins mentioned by Franken but also the water-soluble vitamins, including the antiscorbutic factor.

The first patient treated was a young girl of 17 with advanced pulmonary tuberculosis complicated by intestinal tuberculosis. Seven of her teeth were hypersensitive at the cervix and presented small erosion cavities. The dentin was noticeably soft on the floor of the cavities. Formaldehyde was applied to the hypersensitive areas, but without relief, and the treatment was discontinued. Cod liver oil and tomato juice, together with a liberal quantity of milk, were then prescribed. Two months later, the cervical hypersensitiveness had disappeared in all the affected teeth except the upper left central incisor. This tooth responded to the treatment in about six months. The apparently favorable results here obtained prompted us to prescribe cod liver oil, tomato juice and milk for relief of cervical hypersensitiveness.

Cod liver oil and tomato juice were administered for periods ranging from one to nine months in the twenty patients included in this series. A summary of their progress is presented in the accompanying table. In one, the condition progressed; two were improved; in three, the condition temporarily disappeared but returned in a few months; in four, the cervical hypersensitiveness disappeared in some but not all of the teeth; while eight were entirely free from sensitiveness for periods ranging from six months to two years. Improvement was generally noted in from one to three months. The loss of cervical hypersensitiveness was associated with a definite hardening of the dentin.

Franken prescribes calcium gluconate, a teaspoonful three times a day in orange juice or water, or two tablets three times a day, to combat the mineral deficiency. To my mind, the mineral deficiency can best be corrected by milk, one quart or even more daily. It is interesting to note that all the patients in this series drank little or no milk before their admission to the sanatorium.

CONCLUSION

The use of cod liver oil and tomato juice rich in fat-soluble and water-soluble vitamins is efficacious in the treatment of hypersensitive cervices of teeth. The mineral deficiency may be adequately controlled by the addition of one quart or more of milk daily to the diet. Other observations as yet incomplete suggest that the vitamins here mentioned are also helpful in controlling dental caries.

2. McConkey, Mack: Treatment of Intestinal Tuberculosis with Cod Liver Oil and Tomato Juice, Tr. Nat. Tuberc. A. 25:105, 1929.

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Editorials

THE 1933 MEETING

The long looked for event, the Seventy-Fifth Anniversary of the American Dental Association in conjunction with the Chicago Centennial Dental Congress, convened in the Stevens Hotel, Chicago, August 7-12, and has now become a matter of history. To say that it was a success is merely to reiterate a statement made hundreds of times during the meeting and spontaneously proclaimed at the general sessions, in the section meetings, in the clinics and through corridors of the hotel.

In every way, it was a most stupendous event, and it will be a landmark of dental progress for many years to come. The elaborate preparation presaged its success before the meeting began, and from the day the event was decided on, there has been an amount of energy and acumen displayed that made failure impossible.

The extent and character of the scientific papers combined to

make the meeting from its literary aspect really outstanding, and when the proceedings are issued, it will be found that real progress has been registered. As early as practicable the publication of the papers will begin in THE JOURNAL, and the material will be completed as expeditiously as possible.

There were many distinguished visitors from abroad, to enumerate all of whom would be difficult, but their number and their individual distinction contributed in no small measure to the success of the meeting. Among them might be mentioned Dr. G. Villain, Paris, president of the International Dental Federation; Charles Nord, Holland, secretary of the F.D.I.; A.L.J.C. Van Hasselt, Holland, treasurer of the F.D.I.; Arrigo Piperno, Rome, dentist to Mussolini; A. Dawson Buckley, Nice, president of the American Dental Society of Europe; T. Okumura, Tokio, Japan, dean of the Tokio Dental College; A. L. Hipwell, Paris; Dr. Blase of Greece, Dr. Hess, of Switzerland, Drs. Donahue, Roberts, Griswold and Le Cron, of London, O. Bazant, of Prague, Mrs. Lillian Lindsay, librarian of the British Dental Association, Fred J. Conboy, Toronto, secretary of the Ontario Dental Society; Wallace Seccombe, Toronto, dean of the Royal College of Dental Surgeons, and many other representative men from abroad.

Among the distinguished visitors from this country might be mentioned Surgeon-General Hugh S. Cumming, who has demonstrated the fact that he is a friend of dentistry, and Senator Henrik Shipstead, who before he entered the Senate was a practicing dentist.

This meeting was a demonstration of the real virility of the dental profession. To inaugurate a meeting of this character and magnitude under the handicap of a prolonged industrial depression, and to bring the event to a conclusion as successful as this, was an achievement of no small proportions, and the officers and committeemen have covered themselves not only with distinction but also with actual glory. To particularize would be impossible where so many lent yeoman service to the enterprise, but the four or five hundred men and women who made up the roster of the committees may felicitate themselves on having taken an integral part in one of the great events of dental history. To men and women

of this character depressions do not mean anything except to make them more than ever determined to accomplish their allotted task and force failure out of the way to make room for unbounded success.

The presiding genius of the American Dental Association was G. Walter Dittmar, and of the Congress, Arthur D. Black, both of Chicago. These men devoted themselves to the utmost to the welfare of the meeting, and each of them has an ample reserve of energy, ability and acumen for the promotion and executive consummation of such an enterprise. They should receive the congratulations and hearty thanks of the entire membership.

The attendance was estimated to be between 11,000 and 12,000. The following were elected officers of the American Dental Association: President, A. C. Wherry, Salt Lake City; President Elect, F. M. Casto, Cleveland; Vice Presidents, C. J. Carabello, Tampa, Fla., G. M. Hollenback, Los Angeles, Calif., and H. E. Harvey, Washington, D. C.; Secretary, Harry B. Pinney, Chicago, and Treasurer, Roscoe H. Volland, Iowa City, Iowa.

The next place of meeting will be St. Paul, Minn.

THE CONVENTION: AN INTERPRETATION

The great convention is over and only tremors of its magnificence remain. It took its place near the gates of the Century of Progress and needed not to be ashamed of its habiliments. Wedded art and science displayed themselves for a week and thousands came to see the display. Such was the length and breadth of it that one could not see it or hear it in the time allotted; it was in truth a suburb to the Hall of Science.

Men came and went to rooms and halls to catch a sight or hear a sound. It was all they could do. The effort resembled luring a fish with a line while a school swam by. They sat at times to hear here and wondered what was being said there. Convinced of their inattention at last, they moved away. "Futile!" you say? Far from it. These men who carry the healing of dentistry to far places and often labor in despair will take with them the assurance

that they are a part of a tremendous whole that is slowly, steadily, surely lifting their burdens, lighting their darkness, making more definite the purpose for which they strive. "I could see only a fragment of it," they mutter, "and hear less, but I know it is there. It will come to me."

There is inspiration in that for many a day. Microscopes with arachnoideal eyes stood about; scales that gave molecules conceptual weight; tubes that disclosed parasitic life; colors that gave dress and recognition to ultimate tissues; photographs that put vitamins on parade—all the paraphernalia that mark the age as modern, that bring from their hiding the ultimate causes that in older days were assigned to sorcerers and deities, were there.

Likewise was the work of the artisan's restorative hands. In amazing array, ideas were marshalled in gold and clay, silver and silicate, steel and cement. Some represented the culmination of well tried endeavor, others the beginnings of new departures. Intricate mechanisms and simple ones to serve in their places, costly ones and cheap ones to meet daily needs were close together. Erstwhile problems were simplified and solved by the touch of the master's mechanical hand.

Thus science and art advance against entrenched disease in the name of dentistry. Never before in the history of the profession have talents for pure science and the equally pure applied science been so unified in their purpose.

And more! Two great professions commingled to give steadfastness to their common assault. Physicians and dentists together backed the great issue of health! There was a sort of climax in cooperation manifested here, a cooperation that is daily becoming more necessary and more apparent. No visitor from however distant a place could miss the significance of this.

A final mention. Men were there from foreign lands. They brought reports of their successes and failures, of technics and treatments, of discoveries and further quests. The tang of their speech was the matter of a moment to be lost in the intensity of their purpose—their purpose and ours. Speakers and listeners became enfranchised in the universal democracy of science. They

felt the throb of that high patriotism that is pledged to the highest of high aims, the common well being. Translated, their presence alone meant world wide attack upon the ills that every man there opposed.

T.

NATIONAL BOARD OF DENTAL EXAMINERS

The aim and purpose of the National Board of Dental Examiners will be to establish a standard qualifying dental examination of such character that the Certificate awarded successful candidates may be safely accepted by all State Boards of Dental Licensure as an adequate qualification for the practice of dentistry, subject to the requirements of the laws of the various states; and further, to elevate the standard of qualification for the practice of dentistry.

So much has been said of possible reciprocity in licensing dentists in the different states that anything bearing on this subject is of major interest. The most concrete movement toward establishing a standard qualifying dental examination of such character that the certificate awarded successful candidates may be safely accepted by all state dental boards as an adequate qualification for the practice of dentistry, subject to the requirements of the laws of the various states, was inaugurated when the National Board of Dental Examiners was formed. A body as important and as complicated as this naturally required considerable time for its proper organization, and it is gratifying to know that its personnel is made up of a body of men of outstanding reputation and unusual ability.

In an official communication to THE JOURNAL from Harry W. Nelson, secretary of the national board, we are informed that the first concrete action has just been taken in the form of an examination. We cannot do better than to publish Dr. Nelson's letter in full as a matter of record in a movement of the utmost moment to the welfare of dentistry in this country.

On Friday, May 26 and Saturday, May 27, 1933, the initial Part I examination was given by the National Board of Dental Examiners in four centers, viz., at the Pittsburgh University, Pittsburgh, Pa.; Chicago College of Dental Surgery, Chicago, Ill.; State University of Iowa Dental College, Iowa City, Iowa, and the University of Minnesota Dental School, Minneapolis, Minn.

The Part I examination, embracing the fundamental sciences, consists of written examinations in anatomy, physiology, bacteriology, general pathology, histology and embryology, chemistry and metallurgy. Eighty-three candidates were examined, twenty-nine of whom were from Chicago, twenty-nine from Pittsburgh, seventeen from Minnesota and eight from Iowa. Of the eighty-three candidates, twelve were graduate dentists, fifteen were seniors, thirty-two were juniors, and twenty-four were prejuniors and sophomores.

The personnel of the supervisory boards that conducted the initial Part I examination, May 26-27, 1933, was as follows:

At Pittsburgh University, Pittsburgh, Pa., chief supervisor, W. A. McCready of Pittsburgh, secretary, Pennsylvania State Board of Dental Examiners, assisted by Edward H. Moore, Pittsburgh, and Horace C. Scott, Wilkinsburgh, Pa.

At Chicago College of Dental Surgery, Dental Department of Loyola University, Chicago, Ill., chief supervisor, W. Ira Williams, Chicago, president, Illinois State Board of Dental Examiners, assisted by Clyde H. Warner, Chicago, and Z. W. Moss, Dixon, Ill.

At the State University of Iowa Dental College, Iowa City, Iowa, chief supervisor, John V. Conzett, Dubuque, president, National Board of Dental Examiners, assisted by J. J. Booth, president, Iowa State Board of Dental Examiners.

At the University of Minnesota Dental School, Minneapolis, chief supervisor, Harry W. Nelson, Minneapolis, secretary, National Board of Dental Examiners, assisted by Walter H. Smith, Minneapolis, secretary, Minnesota State Board of Dental Examiners, and Hilen D. Aldrich, St. Paul, member of the Minnesota State Board of Dental Examiners.

The results of the examination were as follows:

Of the eighty-three candidates who took the Part I examination, sixty-eight successfully passed the examination, eight were incomplete, being prejuniors and not having had general pathology as yet, although all eight successfully passed the other five subjects in the Part I examination. Seven failed to pass the examination.

The names of the candidates who finished in the first ten and their averages are as follows: Rufus Ray Beardsley, 1932, Chicago College of Dental Surgery, average 93; Gordon A. Beagle, junior, University of Pittsburgh School of Dentistry, 90.16; Charles G. Stewart, junior, University of Pittsburgh School of Dentistry, 89.5; Theodore R. Mosetich, sophomore, Chicago College of Dental Surgery, 89; Irwin G. Neer, junior, Chicago College of Dental Surgery, 88.67; Henry Langdon Boris, sophomore, Chicago College of Dental Surgery, 88.67; William A. Dickson, 1922, University of Minnesota College of Dentistry, 88.50; Joel Margaretten, junior, University of Minnesota College of Dentistry, 88.50; Charles Ray Parker, junior, University of Pittsburgh School of Dentistry, 88.33; Victor F. Heineman, junior, Chicago College of Dental Surgery, 88.33.

Under the present plans, the National Board of Dental Examiners contemplates holding another Part I examination the first part of December, 1933, and a Part I and II examination in May, 1934.

At the present writing, four states have incorporated into their dental laws sections which provide for the recognition of the National Board of Dental Examiners Certificate under certain specified conditions, viz., Nebraska, Iowa, Pennsyl-

vania and Illinois, and the National Board of Dental Examiners cherishes the fond hope that it may emulate the success of the National Board of Medical Examiners, whose certificate is now accepted by forty-two states.

The significance of this examination must at once be apparent to the entire profession. It is the entering wedge in a movement that has for its object a greater unification in the licensure of the different states. As has so frequently been pointed out, the Federal Constitution grants to individual states a clear cut autonomy in the matter of its internal affairs—its police powers, etc.—leaving no federal provision for universal licensure for professional practice.

The National Board of Dental Examiners has no legal standing. That power must rest with the individual state as one of its sovereign rights. The various state boards will always exist, and it is not the intent or purpose of the National Board ever to supplant them.

Therefore, the inauguration of the National Board of Dental Examiners is a very logical and necessary step toward solving a problem that has confronted our profession from the beginning, and we feel that all of those who are in any way connected with this movement should be heartily congratulated.

The enterprise is akin to that of the Dominion Dental Council of Canada, which has been in operation for years and is doing much toward unifying the professional requirement of the different provinces of that country. In both countries, this movement is worthy of the greatest commendation and encouragement.

THAT FATUOUS FOOTNOTE

In our January, 1933, number, we paid our respects to the Report of the Committee on the Costs of Medical Care, and in particular to a certain footnote on pages 112-13 of the report which said:

> We commend the growing tendency in the practice of dentistry toward a division of labor in which a dentist who is also a physician assumes larger responsibilities for the diagnosis and treatment of conditions arising from or related to the teeth, while much of the routine performed by the dentist in the past is delegated to dental hygienists and other technicians working under his direction.

We took strenuous exception to this assumption at the time, and from other sources have come protests against the accuracy

of the statements made in the footnote, notable among which protests is that contained in an article on pages 81-98 of the *Journal of Dental Research,* February, 1933. Now we have as a recrudescence a "Memorandum from the Dental Educational Council of America" expressing its protest against the misstatements in the footnote, which adds another link to the chain of disapproval that is being welded against this most objectionable document.

Let us comment a moment on some of the circumstances involved in the publication of the footnote. It should be stated that it was signed by seven individuals, only one of whom was a dentist, and he a man who has seldom been conspicuously sympathetic with the dental profession, and not always fully alive to its best interests. All of these seven were supposed to be possessed of discriminating judgment, and at least a few of them had national reputations; but it is apparently a case where little attention was given to the formulation of the statement or else it was delegated to a minority of the committee whose ideas were colored by their own particular bias. In any event, it stands out as an example of warped vision, and unfortunate misinformation. As was pointed out in our editorial of January, it does not represent in any particular the consensus of opinion of the dental profession, either that of the rank and file or that of the most profound student of our major problems.

More than all else, it is a tacit bid for the violation of nearly all of the dental laws of our various states. As a concrete and recent example of this, we have merely to refer to the enactment, at the last session of the legislature of Illinois, of a law which very definitely states what is understood by the practice of dentistry. This clear cut and illuminating statement is noted on page 1504 of the August number of THE JOURNAL, and we commend it to the consideration of every member of the profession. When its provisions are analyzed, it will be apparent how utterly lacking in acumen or in a comprehensive grasp of the real implications of dental practice this unfortunate footnote is. It is authentic neither in fact nor in experience, and we suspect that it was signed by most of the seven individuals without a full understanding of its significance—in fact, we should be surprised if an appreciable percentage of the signers realized what the footnote actually implied.

The Dental Educational Council of America in its "Memorandum" just issued is placing squarely up to the seven signers the alternative of making an explanation of the peculiar ideas advanced in the footnote, or of tacitly acknowledging that they signed it without due deliberation and therefore without warrant. In other words, they must explain or ignore, neither of which alternatives would seem to meet the situation adequately and absolve them of responsibility in the matter. The simple fact is that they cannot explain the erroneous intimations given, and it is doubtful that they will be permitted, by the consensus of professional opinion, to ignore them.

It need not be herein admitted that the signers were for the most part individuals of the highest standing and integrity of character, but a good reputation cannot quite be expected to condone an unfortunate lapse that places on record before the public an erroneous conception of the tendencies in operation in a profession like dentistry. The manly and dignified thing to do would be for the signers either to correct the statements made in the footnote or else to present to the profession any arguments or information that might presumably be supposed to justify those statements.

Obituaries

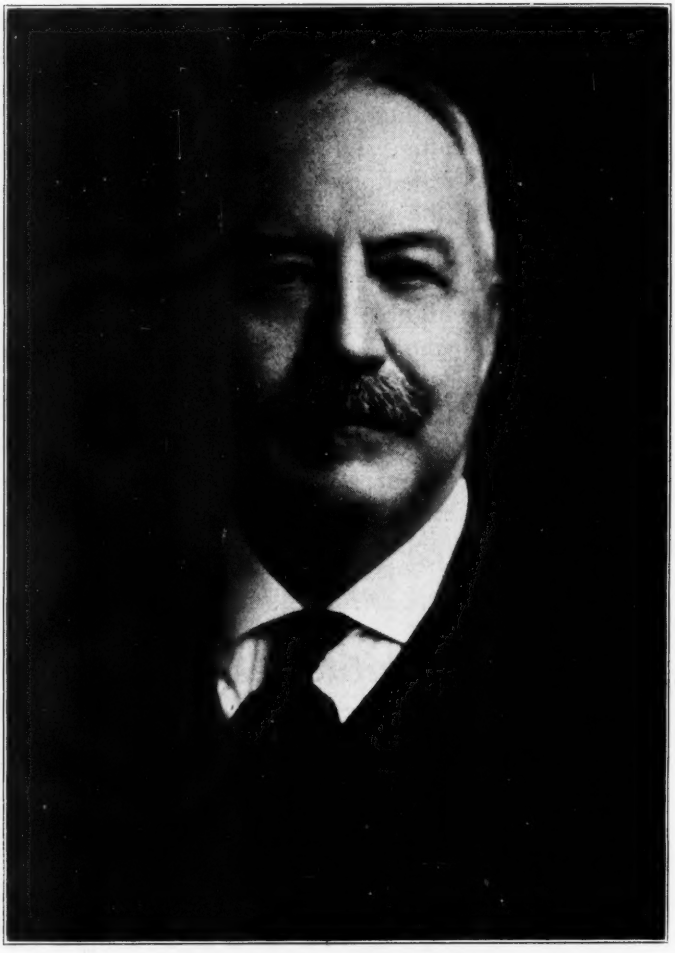
A. HUGH HIPPLE, D.D.S.

(1865-1933)

A. Hugh Hipple was born at Campden, Lincoln Co., Ontario, Canada, Feb. 25, 1865. He met accidental death by drowning while on a fishing trip, near his summer home in Fernbank, Ontario, July 29.

Dr. Hipple received his dental degree from the Royal College of Dental Surgeons at Toronto in 1889. After three years in practice in Stratford, Ontario, he removed to Omaha, Nebr., where he continued his work until the time of his death. He was a member of the American Dental Association and of the American Association of Dental Faculties and a past president of the Nebraska Dental Society and of the Omaha District Dental Society.

In 1908, Dr. Hipple was made dean of the Creighton University Dental School, coming from the medical school of the same institution, where he had been an



A. HUGH HIPPLE, D.D.S.
(1865-1933)

instructor in oral surgery for thirteen years. He was actively identified with the work of the School for the Deaf, being on its board of directors. He was a member of the Methodist Episcopal Church and had many club and filial associations.

In 1896, Dr. Hipple married Miss Emma Pritchard, of Brockville, Ontario, who survives him, together with one daughter, two grandchildren, a brother and two sisters.

Interment was in Brockville.

EDWARD CAMERON KIRK, D.D.S., Sc.D., LL.D.

(1856-1933)

Edward Cameron Kirk was born in Sterling, Ill., Dec. 9, 1856; died, July 20.

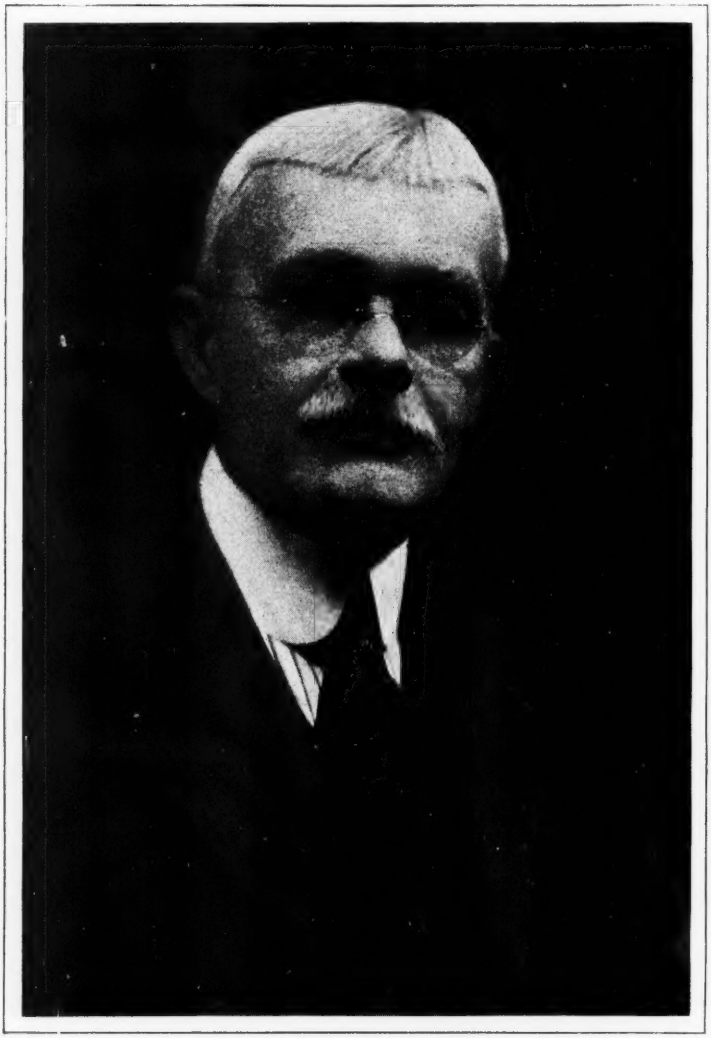
After a thorough elementary education, followed by an assistantship in the chemical department of the Towne Scientific School of the University of Pennsylvania, Dr. Kirk entered on the study of dentistry in the Medical Department of the University of Pennsylvania. The following year, he transferred to the Pennsylvania College of Dental Surgery, from which he was graduated in 1878. From that year also dates his connection with the dental department of the university, of which he was the executive officer from 1896 to 1917. He was appointed demonstrator in prosthetic dentistry during the first session of the school. In 1882, he was appointed to the lectureship in operative dentistry and he later became professor of clinical dentistry.

His ability as a writer is evinced by his numerous contributions on practically every subject in the field of dentistry. In 1891, he became editor of the *Dental Cosmos,* continuing in that capacity for thirty-nine years. He contributed chapters to the "American System of Dentistry" and edited the "American Textbook of Operative Dentistry," to which he contributed several articles.

In 1903, Dr. Kirk received the honorary degree of doctor of science and in 1915 the honorary degree of doctor of laws. He received several French decorations in recognition of his interest in dental science and education.

Dr. Kirk was a past president of the Pennsylvania State Dental Society and the Academy of Stomatology and a member of the American Dental Association.

In July, 1917, he resigned from the active teaching force of the University of Pennsylvania to enter the service of the S. S. White Dental Manufacturing Co., of which he became vice president.



EDWARD C. KIRK, D.D.S., Sc.D., LL.D.
(1856-1933)

COUNCIL ON DENTAL THERAPEUTICS

ACCEPTED DENTAL REMEDIES

The following articles have been accepted as conforming to the rules of the Council on Dental Therapeutics of the American Dental Association for inclusion in the list of Accepted Dental Remedies. The Council desires dentists to understand that the admission of an article does not imply a recommendation. A copy of the rules which govern the Council in the consideration of articles will be sent on request.

SAMUEL M. GORDON, *Secretary.*

VIOSTEROL IN OIL 250 D-ABBOTT.—A brand of viosterol in oil 250 D, N.N.R.

Manufactured by The Abbott Laboratories, North Chicago, under U. S. patent 1,680,818 (Aug. 14, 1928; expires 1945) by license of the Wisconsin Alumni Research Foundation.

Actions and Uses: See Viosterol, THE JOURNAL, 18:1786 (Sept.) 1931.

Dosage: See Viosterol, THE JOURNAL, 18:1786 (Sept.) 1931. Viosterol in oil 250 D-Abbott is prepared by dissolving ergosterol in anhydrous, peroxide-free ether, U.S.P. The solution is filtered, placed in transparent quartz containers with reflux condensers and exposed to ultraviolet rays at a determined distance and intensity for a determined length of time. The irradiated ergosterol, freed of ether and dissolved in sesame oil, is biologically assayed and adjusted to have the potency of viosterol in oil 250 D, N.N.R.

ABBOTT'S COD LIVER OIL WITH VIOSTEROL 10-D.—A brand of cod liver oil with viosterol 10 D, N.N.R.

Actions and Uses: See Cod Liver Oil with Viosterol, THE JOURNAL, 18:1787 (Sept.) 1931.

Dosage: See Cod Liver Oil with Viosterol, THE JOURNAL, 18:1787 (Sept.) 1931.

Manufactured by The Abbott Laboratories, North Chicago, under U. S. patent 1,680,818 (Aug. 14, 1928; expires 1945) by license of the Wisconsin Alumni Research Foundation.

Irradiated ergosterol, prepared by the method described under viosterol in oil 250 D-Abbott, is added to cod liver oil and the finished product is required to have a vitamin A potency of not less than 500 pharmacopeial units per gram and to have the vitamin D potency of cod liver oil with viosterol 10 D, N.N.R.

DETROIT DENTAL AID COMMISSION TOOTH POWDER.—The product is stated to be made up as follows: calcium carbonate, precipitated, 60 gm.; magnesium oxide, 2 gm.; magnesium carbonate, 1 gm.; sodium bicarbonate, 30 gm.; powdered Castile soap, 6 gm.; sodium chloride (finely milled), 5 gm.; saccharine, 0.2 gm.; oil of Gaultheria synthetic, 72 minims; oil of peppermint, 8 minims.

Claims: The Commission states that no claims will be made for this preparation other than of cleaning the teeth when used with the toothbrush.

Distributed by the Detroit Dental Aid Commission of the Detroit District Dental Society, Detroit, Mich. No U. S. patents or trademarks.

PARKE-DAVIS DENTAL CREAM.—Composition: Each hundred grams is stated to contain magnesium hydroxide, not less than 3.7 gm.; glycerine, 31.3 gm.; corn starch, 4.7 gm.; saccharine, soluble, 0.015 gm.; calcium carbonate, precipitated, 46.9 gm.; soap, 2.3 gm.; flavoring, 0.70 gm. (oil of peppermint, oil of spearmint, oil of anise, menthol); water, 10.44 gm.

Claims: No claims, except that it is an agent to be used for cleaning the teeth when used in conjunction with the toothbrush, are made.

Manufactured and distributed by Parke, Davis & Company, Detroit, Mich. No U. S. Patent or trademark.

CURRENT LITERATURE

Agranulocytic Angina—Treatment by the Use of Parenteral and Oral Liver Extract: Preliminary Report (F. L. Foran, H. M. Sheaff and R. W. Trimmer, J.A.M.A., June 17, 1933, p. 1917): This syndrome was described by Schultz under the term agranulocytic angina. It is otherwise designated as malignant neutropenia and pernicious leukopenia. No highly satisfactory treatment has as yet been demonstrated for it. The mortality of this disease when untreated is above 90 per cent. When blood transfusions have been employed, the mortality is 64 per cent; that following roentgen irradiation is 53 per cent. Leukopoiesis following the use of liver extract had been observed before this report. The fact that the disease is characterized by a defective function of the bone marrow furnishes the basis for the liver treatment. The intravenous route was preferred for acute cases. Four reports are made. All four patients recovered, one to die of pneumonia later. The other three remained clinically and hematologically well.

T.

Progress in Anesthesia in 1932 (R. F. Sheldon, New England J. Med., June 29, 1933, p. 1369): Dr. Sheldon reviews the usage of anesthetics, giving a particularly interesting discussion of avertin. He quotes its users. Bourne says, "There are no contraindications to the use of this bromin substitution product of ethel alcohol provided its dosage suits the case"; Balaam says, "Avertin is as far ahead of these (six other commonly used anesthetics) as a motor car is ahead of a bicycle"; Paterson and Pierce say "It irritates none of the body organs in either its administration or its elimination." Some German authors assert that it is a liver poison. Ashworth reported three cases in which there were no ill-effects on the liver.

T.

Syphilis in Relation to Cancer of the Buccal Mucosa (Charles Lund, New England J. Med., July 21, 1933): The article begins with the clinical diagnosis of both syphilis and cancer and concludes that when the diagnosis of cancer or gumma cannot be definitely made on clinical ground at an early stage, it is obvious that, when the two diseases coexist, the clinical diagnosis will be more difficult. Some cases of cancer arise with syphilis without syphilis being the predisposing cause. The opposite also occurs. Again, both diseases may be active in the same lesion. This last condition seems to be rather rare. Charts are given presenting the incidence of the foregoing possibilities. Following are the summary and conclusions: 1. The differential diagnosis of syphilis of the mouth, carcinoma of the mouth and combined syphilis and carcinoma of the mouth is difficult and can be made surely only by pathologic examination. 2. In this cancer clinic, cancer of the mouth was found twelve times more frequently in patients with evidence of syphilis or positive Wassermann reactions than purely syphilitic lesions of the mouth were found in such patients. 3. Syphilis is definitely an agent contributing to the cause of cancer of the tongue in a large number of cases (about one-quarter), but it is a much less important etiologic agent in cancer of all other parts of the mouth. 4. One-half of the patients wih cancer and syphilis were treated for weeks or months for syphilis before coming to the clinic. Patients with syphilis and cancer of the tongue whose lesions were studied to determine the pathologic index of malignancy showed a higher proportion of patients with very malignant tumors than patients without syphilis. Statistical chance and these two factors working alone or in any combination can account for the apparently poor results seen in the treatment of these cases in this series.

T.

Pathology of Carcinoma of the Buccal Mucosa in Relation to Results of Treatment (Charles Lund, New England J. Med., July 20, 1933): Dr. Lund quotes the Cancer Commission of Harvard University, Broders, Helwig, Warren and Simmons, and writes under the heads: epidermoid carcinoma, pathologic index of malignancy, index in relation to location of tumor, size of tumor and relation of duration to pathologic index. He

gives eight tables from the authors mentioned including lip and tonsil. He summarizes as follows: 1. Studies of the pathologic index of malignancy of carcinoma of the mouth have been made by two observers and compared with the results of treatment of the cases. Although the classifications differed, there was definite value in both. 2. Proportionately, carcinoma of the tongue showed a higher index of malignancy than carcinoma elsewhere in the mouth. The results of treatment were equal in cases of a given index, irrespective of location except for carcinoma of the lip. 4. The importance of the pathologic index of malignancy as one factor to be considered in the complete study of carinoma of the mouth is reaffirmed.

T.

Surgical Treatment of Trigeminal Neuralgia (Adrian S. Taylor, Am. J. Surg., June, 1933): The author notes an abundant literature on the subject and that the etiology and pathology of the condition are unknown. He states that high blood pressure and arteriosclerosis as a cause deserves further study. Although tic douloureux is a disease of the fifth cranial nerve, there is also the fairly well-known tic of the glossopharyngeal nerve which is more terrible, if that is possible. Tic douloureux should be differentiated from Sluder's neuralgia, which may be relieved by cocainization of Meckel's ganglion or the removal of local foci of infection. Futile attempts are made to aid patients with true tic by the removal of foci, and few patients reach the surgeon who have not had some teeth removed. The author has seen no patients relieved by these operations. Dandy has perfected his cerebellar approach until he is able to accomplish his operation in a fraction of the time used in the transtemporal operation. The cerebellar approach should not be undertaken by one unskilled in posterior fossa surgery. The author describes the various injection procedures, but says that the radical operation has been so systematized that it has become a relatively simple procedure.

T

FOREIGN

Utilization of Nature's Articulator in Denture Construction (D. Stuart MacKenzie, New Zealand D. J., May, 1933, p. 164): With full respect paid to artificial articulator advocates, MacKenzie proclaims the jaw itself to be the best possible articulator. He has unraveled a method which he has followed for twelve years and which, because of its results, has led him to suspect errors in the widely taught usages of the ingenious devices of Bonwell, Gysi *et al.* He doubts that anatomic articulators should be modeled on the movements of the perfectly functioning jaw. Such movements are incapable of standardization. He has found that it is much easier to make dentures if he does not extract all the teeth at once but adds to his dentures as he extracts, leaving the incisors to the last. In this manner, he reproduces nature's esthetics. Measurements are made and recorded while the teeth are in normal occlusion—the key to the whole process. After absorption is complete, the measurements maintained become of paramount importance. Patients are advised to visit the surgery every twelve months to have absorption checked up. Even so slight a change as 2 mm. is corrected.

T.

Paradentosis (Dr. Adrion, Deutsche Med. Wchnschr., 59:879-880, 1933): The author defines paradentosis as a disease of the entire masticatory apparatus, e.g., the teeth, peridental membrane, alveolar bone and mouth mucosa. They are all functionally interdependent, influencing one another. It is caused by both exogenous and endogenous hereditary conditions which result in a slowly developing and painless affliction. It begins as a superficial inflammation of the gums which leads to paradental tissue and bone destruction, accompanied by inactive granular tissue formation in the peridental space. The clinical manifestations are light bleeding from the gums, fetid odor, occasional flow of pus on pressure, single or multiple loosening and loss of teeth, and torsoclusion of an anterior tooth. The following three forms are often represented clinically: Catarrhal: The gums bleed and appear swollen, with a bluish or dark red margin. The papillae are edematous and thickened. This form may result in loosening of teeth and catarrhal paradentitis, with pocket formation. The epithelium in this pocket is pathologically changed and presents the picture of granular tissue. Hyperemia develops around the seat of infection and hinders the regular supply and drainage of blood and other secretions. Hypertrophic: This form is represented by a paradental abscess arising from the accumulation of the foregoing secretions. Atrophic: The gums shrink and recede, without pocket formation. The loosening of the teeth is caused by the

absorption of the alveolar bone, by the widening of the peridental space and by replacement of the peridental membrane with granular tissue. The treatment consists of prophylaxis, thorough scaling of all teeth and a thorough care of the mouth by the patient. The use of medicines is not indicated. The inflammation should be treated as such, and its causes removed.

OSCAR KARPF.

Bacteriology of Gangrenous Root Canals and Effects of Different Dental Germicidal Remedies (Maximilan Fisher Zahnärtzl. Rundschau, 42:1039-41, 1086-1090, 1933): In the treatment of infected dental pulps, a method is to be aimed at that will not only render the aerobic bacteria harmless, but also, first, destroy the anaerobes. With present-day methods, it is not always possible to accomplish this with the spore forming anaerobes. Experiments are in progress to discover effects of the use of general antiseptics on anaerobic bacteria and to determine a method by which it will be possible to eliminate the bacteriologic danger. An antiseptic prohibiting bacterial growth is not satisfactory since it is of importance to destroy the spores as well. The author admits that this involves extreme difficulty. He believes that no other method than "fractional sterilization" is available. This involves the destruction of the vegetative forms first, with subsequent elimination of the spores by suitable methods. The Fisher Kruse silver reaction process promises results in this direction, especially when used in combination with diathermy.

OSCAR KARPF.

Secondary Formation of Odontoblasts (Herman, Euler, and H. H. Rebel, Ztschr. f. Stomatol., 30:515-530, 588-600, 1932): The author shows, with the aid of preparations from fractured teeth, free denticles and, from experimentally produced trauma, secondary formation of odontoblasts. They are formed as a result of differentiation of undifferentiated mesenchymal cells of the dental pulp by virtue of its regenerative powers. Findings in experimental amputations and extirpations on monkeys showed that the growth of the root does not stop after the destruction of the primary odontoblasts. This growth is accomplished both through the formation of a bonelike structure and through the formation of new odontoblasts and secondary dentin. The practical importance of pulp treatment may be emphasized by these findings.

OSCAR KARPF.

Ueber die biologischen Vorgänge an amputierten. Pulpen (J. Münch, Ztschr. f. Stomatol., 30-1457, 1932): The author believes that the dictum that each diseased pulp is a lost organ is not justifiable as long as one does not expect the remainder of the amputated pulp to maintain its original function. By means of strict asepsis and special amputation technic (local anesthesia, placement of calcium and vitamin-containing preparations on the amputation stump), it is possible to institute biologic processes in partially diseased pulps. These processes lead by means of increased secondary dentin formation to a complete or partial closing of the root canals. A spontaneous biologic root filling may thus be achieved. It is also possible that the pulp tissue or the contents of the root canal may, by calicification, become passively obliterated. Age, the vitality of the pulp and the general disposition play an important rôle in conditioning these processes.

I. S.

Histologische Untersuchungen über die Heilung von Extraktionswunden nach der chirurgischen Entfernung der Fazialen Alveolarwand (Karoly Balogh, Ztscher. f. Stomatol., 30:281, 1932): Experimental studies conducted on dogs showed that the healing process of the extraction wound following the removal of the labial alveolar bone corresponded with that found in normal extraction wounds. The only difference lies in the fact that the particular phases of healing are shorter in duration. This can be explained on the basis that the margins of the wound are united by suture and that the wound cavity is made smaller by the removal of the labial plate. The author found in no case a secondary wound infection, which is not so rare in open extraction wounds. According to Euler, the wound healing starts from the gingival tissues. This circumstance is beneficial in the wounds found in the investigation, because they are covered by a greater gingival surface. Thus, the wound healing occurred faster in the experiments than in normal cases. One could not speak of complete healing until after twelve weeks.

I. S.

Induction Anesthetics, Especially on Pernocton (Yoshimi Matsuda, Rinsho Shika): The author discusses induction anesthesia, especially pernocton, in the field of dentistry.

Following are the conclusions. 1. Pernocton provides the properties which are desirable in induction anesthesia for oral surgery and in other fields of dental practice. 2. In case of an intravenous injection, a rapid action will be produced. A slow injection is said to be more effective for a mild action of this anesthetic. 3. The rapidity of injection is particularly important, consequently careless injection is absolutely prohibited. One or two minutes per cubic centimeter should be employed. The author uses two minutes. 4. The quantity employed is 1 c.c. per 12.5 kilograms of body weight. It is needless to explain that there exist some differences in consideration of a well or poorly developed physique. A safe procedure is to stop at the point where the patient becomes sleepy. 5. The duration of anesthesia may be more or less different in accordance with individual physique as well as quantity of injection. 6. The quantity for anesthesia will not result in damage particularly on the vasomotor nerves and respiration center. An increase of the pulse rate will temporarily be brought about. It usually disappears soon after. 7. It is wise to employ it cautiously particularly in cases of heart disease and in children under 15 years. 8. Overdosage of pernocton is absolutely contraindicated. 9. Aftereffects may sometimes be present. Vomiting sometimes appears, yet this can be prevented by means of atropin injection or injecting the anesthetic solution slowly. 10. Continuously repeated injection should be avoided. 11. The aim should be simple sleep. No disaster will occur from the use of this anesthetic.—Abstr. in *Rinsho Shika* (English edition), May-June, 1933.

Oral Tuberculosis (Jinzaburo Yamagami, Rinsho Shika): In summing up, the author states that tuberculosis is usually curable, especially when diagnosed early. Neuman's operation for pyorrhea or difficult extraction of a horizontally erupted wisdom tooth should be avoided if a patient has a predisposition to tuberculosis. With regard to the procedures, a systemic therapy should be referred to a physician. Roentgen-ray radiation and ultraviolet ray therapy are only to be employed as supplemental treatment. In a word, the disease focus must be radically removed.—Abstr. in *Rinsho Shika* (English edition), May-June, 1933.

MISCELLANY

BOOK REVIEWS

The Operative Story of Cleft Palate. By George Morris Dorrance, M. D. Published by W. B. Saunders Company, Philadelphia.

The "Operative Story" comprises 442 pages of history reaching from 460 B.C. to the present time. Within that compass are 522 illustrations of instruments, technics and conditions. An appendix of six pages of illustrations is added. In addition, eighty-seven pages are devoted to bibliography. Every phase of the subject is presented and discussed to completion. It is an exhaustive work, a library of information in itself. Teachers of oral surgery will find it a reference book without a peer. It is a summation of the knowledge and experience, the observation and deduction of a great worker and a noted surgeon.

The Duties of the Ohio Public Health Commissioners. By W. W. Charters and D. A. Hindman, Ohio State University Studies No. 17.

Following the appointment by the president of a committee to study the problem of reorganization of the courses offered by the Ohio State University in health education, the committee became interested in the training of public health officials. Since the state requires public health commissioners the committee deduced that programs to fit men for such offices might be required of the university. Accordingly, a subcommittee was appointed for the purpose of investigating curriculums for public health commissioners. A necessary basis for such a curricular provision was perforce a detailed knowledge of the duties that fell to such officials. Such an analysis had never before been made. The university did not contemplate the installation of undergraduate courses, but short courses based upon the analyses of the committee. From them, courses for prospective commissioners might be made later. A study of the document developed by the subcommittee shows the tremendous scope of the public health

officials' work and that a medical training cannot guarantee fitness for it. After the compilation of duties had been made and studied, specifications for courses were submitted. Likewise, after the survey of the activity lists, definite units were prescribed. The study constitutes an invaluable guide for other localities and schools aroused to the same need and undertaking a similar project.

"AN APPEAL FOR MENTAL FOOD"

The following letter is addressed "to the American authors of dental books, to publishers of same and to publishers of dental magazines":

Gentlemen:

The Students' Association of the Dental School of the University of Athens, Greece appeals to you for mental food. We are hungry for knowledge and we haven't any money to buy your books and magazines. The Sons of the Old Glory That Was Greece want you to give them a helping hand.

We rely on your good heart to mail us a copy of each of your dental publications for our student library. Good hearted fellows, please respond to our call and mail literature to: The Students' Association of the Dental School, University of Athens, 3 Sina Str., Athens, Greece. G. SPANOS, *President.*

NEWS

DEATHS

Fitch, Clyde E., Laramie, Wyo.; Creighton University, College of Dentistry, 1925; died, July 18, 1932.

Peterson, Hugo E., Rawlins, Wyo.; School of Dentistry, University of Denver, 1918; died, March 14, 1933.

Tisch, Henry E., Wheatland, Wyo.; School of Dentistry, University of Denver, 1903; died, April 7, 1933.

ANNOUNCEMENTS*

CALENDAR OF MEETINGS

American Board of Orthodontia, Oklahoma City, Okla., November 6-7.

American Dental Society of Europe, Holland, August, 1934.

American Society of Orthodontists, Oklahoma City, Okla., November 8-10.

Greater New York December Meeting, New York City, December 4-8.

International Congress of Radiology, Zurich, Switzerland, July 24-31, 1934.

Odontological Society of Western Pennsylvania, Pittsburgh, November 14-16.

Society for Advancement of General Anesthesia in Dentistry, New York City, third Monday in February, April, October and December.

Montreal (Canada) Dental Club Annual Fall Clinic, October 12-14.

District of Columbia, at George Washington University, Washington, D. C., second and fourth Tuesdays in each month from October to June, inclusive.

*Announcements must be received by the fifth day of the month in order to be published in the forthcoming issue of THE JOURNAL.

STATE BOARD OF DENTAL EXAMINERS

California, at San Francisco, December 11. K. I. Nesbett, Secretary, State Bldg. Annex, San Francisco.

Indiana, at Indianapolis, October 30. J. M. Hale, Secretary, Mt. Vernon.

MICROSCOPIC, ORAL CAVITY AND BONE DIAGNOSTIC DEMONSTRATION

A microscopic, oral cavity and bone diagnostic demonstration, with lantern slides, will be held at the Mayflower Hotel, Washington, D. C., September 17-24. Pathologists, radiologists, surgeons, dentists and physicians interested in the diagnosis and treatment of cancer in all stages and in the local conditions that precede cancer are especially urged to attend. Artificially cooled demonstration rooms are furnished by the hotel without cost. Single rooms with bath are available at $3.50 per person; large double rooms with twin beds and bath, $2.50 per person. Address Mr. Pollio for reservations. The demonstration cannot be definitely scheduled until 100 acceptances have been received. Anyone who wishes to present a case at any of the demonstrations should notify Dr. C. F. Geschickter, Johns Hopkins

Hospital, Baltimore, Md. Sunday evening, September 17, there will be a conference on the problem of preoperative irradiation; Monday, a microscopic lantern slide demonstration; Tuesday to Thursday, the regular bone demonstration; Friday-Saturday, oral cavity demonstrations. Address acceptances to

JOSEPH COLT BLOODGOOD,
3301 N. Charles St.
Baltimore, Md.

GREATER NEW YORK DECEMBER MEETING

The ninth annual Greater New York December Meeting will be held at the Hotel Pennsylvania, New York City, December 4-8, under the auspices of the First and Second District Dental Societies of the State of New York.

CARROLL B. WHITCOMB, *Chairman.*

ODONTOLOGICAL SOCIETY OF WESTERN PENNSYLVANIA

The fifty-second annual meeting of the Odontological Society of Western Pennsylvania will be held at the William Penn Hotel, Pittsburgh, November 14-16.

W. EARLE CRAIG, *Secretary,*
Jenkins Arcade
Pittsburgh, Pa.

INDIANA STATE BOARD OF DENTAL EXAMINERS

The next meeting of the Indiana State Board of Dental Examiners for examination of applicants for licensure in Indiana will be held in Indianapolis, beginning Monday, October 30, and continuing four or five days. Applications and all proper credentials together with necessary fee must be in the hands of the secretary at least five days prior to the date of the examination. For instructions to candidates, applications and further information, address

J. M. HALE, *Sec'y-Treas.,*
Mt. Vernon.

AMERICAN SOCIETY OF ORTHODONTISTS

The thirty-second annual meeting of the American Society of Orthodontists will be held at Oklahoma Biltmore Hotel, Oklahoma City, November 8-10. All members of the American Dental Association who are recommended by a member of the American Society of Orthodontists are welcome to attend the sessions; registration fee for nonmembers, $10. Hotel rooms with bath are available at $2.50 a day. Write for reservations. Golf and side trips to the Gulf of Mexico are a part of the program of entertainment.

CLAUDE R. WOOD, *Secretary,*
Medical Arts Bldg.
Knoxville, Tenn.

MONTREAL DENTAL CLUB NINTH ANNUAL FALL CLINIC

The Ninth Annual Fall Clinic of the Montreal Dental Club will be held October 12-14, at the Windsor Hotel. Fee, including two luncheons, $15; for those registering from points outside the City of Montreal, $7. Register with A. R. Winn, 1240 Union Ave., Montreal. For exhibit space, address A. D. Richardson, 1240 Union Ave., Montreal.

H. R. CLEVELAND,
Chairman of Publicity,
1240 Union Ave.
Montreal.

IDENTIFICATION SOUGHT

The State Highway Department of Delaware requests publication of the following description of teeth of woman whose nude body was found floating in the Delaware River, near Wilmington, Del., July 10, 1933: All upper teeth missing, lower two central and two lateral incisors, two cuspids, right bicuspid and second molar intact; incisal edge of anterior teeth abraded, indicating the age of the woman to be between 35 and 40 years.

Address information to:

C. C. REYNOLDS, *Supt.,*
State Highway Dept.,
Odd Fellows Bldg.,
Wilmington, Del.

RECENT BOOKS WORTH BORROWING* OR BUYING†

from the

LIBRARY BUREAU

of the

AMERICAN DENTAL ASSOCIATION

212 E. Superior St., Chicago, Ill.

Adams, C. W.	Your teeth and their care. St. Louis, Mosby, 1932. $1.25. Alfred David Lenz system of lost wax casting. New York, National Sculpture Soc., 1933. $1.50.
American Pharmaceutical Assn.	Pharmaceutical recipe book. Baltimore, Md., American Pharmaceutical Assn., 1929. $5.00.
Anderson, G. M.	Orthodontic laboratory manual. St. Louis, Mosby, 1930. $2.00.
Anthony, L. P.	Dental terminology. Chicago, American Dental Assn., 1930. (Free.)
Appleton, J. L. T., Jr.	Bacterial infection with special reference to dental practice. Philadelphia, Lea & Febiger, 1925. $6.00.
Appleton, J. L. T., Jr., & Bryant, C. K.	Laboratory guide in bacteriology. Philadelphia, Lea & Febiger, 1928. $2.75.
Bartels, H. A.	Outline of bacteriology. New York, W. A. Broder, 1929. $2.00.
Beck, Dorothy F.	Cost of equipping a dental office. Chicago, University of Chicago Press, 1932. $1.00.
Berger, Adolph	Principles and technique of oral surgery. Brooklyn, Dental Items of Interest Pub. Co., 1927. $7.00.
Berger, Adolph	Principles and technique of the removal of teeth. Brooklyn, Dental Items of Interest Pub. Co., 1929. $7.50.
Blunt, Katharine, & Cowan, Ruth	Ultraviolet light and vitamin D in nutrition. Chicago, University of Chicago Press, 1930. $2.50.
Bödecker, C. F.	Elementary histology for dental hygienists. New York, W. A. Broder, 1929. $2.00.
Bogert, L. J.	Nutrition and physical fitness. Philadelphia, Saunders, 1931. $3.00.
Bosworth, H. J.	Dental economics. Chicago, Bosworth Economic Institute, 1932. $10.00.
Bowler, Inez	Elementary manual of dental library practice. Ann Arbor, Univ. of Michigan Press, 1932.
Box, H. K.	Necrotic gingivitis. Toronto, University of Toronto Press, 1930. $1.00.
Box, H. K.	Treatment of the periodontal pocket. Toronto, University of Toronto Press, 1928. $1.00.
Bregstein, S. J.	Business conduct of an ethical practice. Brooklyn, Dental Items of Interest Pub. Co., 1932. $4.00.
Broderick, F. W.	Dental medicine. St. Louis, Mosby, 1928. $6.00.
Broderick, R. A.	Dental bacteriology. Outlines of dental science. Vol. 2. Edinburgh, Livingstone, 1926. $2.50.

*Books may be *borrowed* by members of the Association for a period of one week after date of receipt; and if not reserved for some one else, the period will be extended on request. A deposit of $2.00 for each volume to be sent at one time should accompany all requests for books. This will be refunded on return of books.

†If you wish to *purchase* books, indicate plainly which books you desire and enclose check with order.

Brothers, E. D.	Dental jurisprudence. St. Louis, Mosby, 1928. $2.25.
Brown, A. J.	Dental metallurgy. Outlines of dental science. Vol. 11. New York, Wood, 1928. $2.50.
Brown, W. L.	Endocrines in general medicine. St. Louis, Mosby, 1927. $3.00.
Buckley, J. P.	Modern dental materia medica, pharmacology and therapeutics. Philadelphia, Blakiston, 1926. $6.00.
Bulleid, Arthur	Textbook of bacteriology for dental students. St. Louis, Mosby, 1927. $4.50.
Bunce, E. W.	Bunce-Kanouse full denture technic. Chicago, Coe Laboratories, Inc., 1929. $1.25.
Bunting, R. W.	A textbook of oral pathology for students and practitioners of dentistry. Philadelphia, Lea & Febiger, 1929. $7.00.
Burchard, H. H., & Inglis, O. E.	Textbook of dental pathology and therapeutics for students and practitioners. Philadelphia, Lea & Febiger, 1926. $7.50.
Burkhart, H. J.	Care of the mouth and teeth. (National Health Series.) New York, Funk & Wagnalls, 1928. $0.30.
Buxton, J. L. D.	Dental pathology. Outlines of dental science. Vol. 7. New York, Wood, 1927. $2.50.
Buxton, J. L. D.	Dental surgery. Outlines of dental science. Vol. 8. New York, Wood, 1927. $2.50.
Cahn, L. R.	Basic principles of general and oral pathology. Brooklyn, N. Y., Dental Items of Interest Pub. Co., 1929. $4.50.
Campbell, Menzies	Those teeth of yours. London, Heinemann, 1931. $1.50.
Casto, T. D.	Questions and answers on pedodontology, with glossary. Philadelphia, Author, 1932. $2.50.
Cattell, Psyche	Dentition as a measure of maturity. Cambridge, Harvard University Press, 1928. $1.00.
Churchill, H. R.	Human odontography and histology. Philadelphia, Lea & Febiger, 1932. $4.50.
Clark, C. A.	Dental radiography. Outlines of dental science. Vol. 5. New York, Wood, 1926. $2.50.
Cogswell, W. W.	Dental oral surgery. Pittsburgh, Penna., Dental Digest, Inc., 1932. $10.00.
Colyer, J. F., & Sprawson, Evelyn	Dental surgery and pathology. New York, Longmans, 1931. $12.50.
Colyer, Stanley	Chronic infection of the jaws: a short radiological and clinical study. New York, Macmillan, 1926. $3.50.
Committee on Costs of Medical Care	Medical care for the American people. Chicago, Univ. of Chicago Press, 1932. $1.50.
Dall, William	Ground porcelain inlays. London, Henry Kimpton, 1933. $5.25.
Daukes, S. H.	Medical museum: Modern developments, organization and technical methods based on a new system of visual teaching. London, Wellcome Foundation, Ltd., n.d. (No purchase orders accepted.)
Davis, M. M.	Paying your sickness bills. Chicago, University of Chicago Press, 1931. $2.50.
Dental Board of the United Kingdom	Four lectures on "general anaesthesia." London, Dental Board of the United Kingdom, 1926. $1.25.
Dental Board of the United Kingdom	Three lectures on "dental alloys." Delivered under the auspices of the Dental Board of the United Kingdom. London, Dental Board of the United Kingdom, 1927. $1.25.
Diamond, M.	Dental anatomy. A graphic presentation of tooth forms with an original technique for their reproduction. New York, Macmillan, 1929. $5.00.
Dorrance, G. M.	Operative story of cleft palate. Philadelphia, Saunders, 1933. $6.50.
Doxtater, L. W.	Procedures in modern crown and bridge-work. Brooklyn, N. Y., Dental Items of Interest Pub. Co., 1931. $8.00.

Endleman, Julio	Special dental pathology; a treatise for students and practitioners. St. Louis, Mosby, 1927. $7.00.
Ennis, L. M.	Dental roentgenology. Philadelphia, Lea & Febiger, 1931. $6.50.
Farkasch, O.	Dilemmas of dentistry. New York, Dodd Bros., 1931. $3.50.
Federspiel, M. N.	Harelip and cleft palate. St. Louis, Mosby, 1927. $5.00.
Felcher, F. R.	Art of porcelain in dentistry. St. Louis, Mosby, 1932. $9.00.
Feldman, M. H.	Manual of exodontia. Philadelphia, Lea & Febiger, 1930. $3.50.
Ferguson, H. W.	Child's book of the teeth. Yonkers-on-Hudson, World Book Co., 1932. $0.68.
Fine, Samuel	Principles and practice of dental assistant. Worcester, Mass., Hamilton Pub. Co., 1931. $2.50.
Fish, E. W.	Experimental investigation of enamel, dentine and the dental pulp. London, John Bale, Sons & Danielsson, Ltd., 1933.
Fones, A. C.	Mouth hygiene; a textbook for dental hygienists. Philadelphia, Lea & Febiger, 1927. $5.00.
Fones, A. C.	Preventive dentistry. Philadelphia, Lea & Febiger, 1925. $2.75.
Foote, J. S.	Bone as a measure of development: When and how we acquired our teeth. Chicago, American Dental Association, 1928. $4.00.
Franzen, Raymond	Public health aspects of dental decay in children. New York, American Child Health Assn., 1930. $1.25.
Gies, W. J.	Dental education in the United States and Canada. New York, Carnegie Foundation, 1926. (Free.)
Gillett, H. W., & Irving, A. J.	Gold inlays by the indirect system. Brooklyn, Dental Items of Interest, 1932. $8.00.
Goepp, R. M.	Dental state board questions and answers. Philadelphia, Saunders, 1928. $4.50.
Goslee, H. J.	Principles and practice of crown and bridge-work. Brooklyn, Dental Items of Interest Pub. Co., 1926. $7.50.
Gottlieb, B., & Orban, B.	Die veränderungen der gewebe bei übermässiger beanspruchung der zähne. Leipzig, Georg Thieme, 1931. Unbound, $6.50. Bound, $7.00.
Graves, Lulu G.	Foods in health and disease. New York, Macmillan, 1932. $3.50.
Greenfield, A. L.	Interpretation of dental radiograms. Rochester, N. Y., Ritter Dental Mfg. Co., 1926. $2.50.
Gregory, W. K.	Our face from fish to man. New York, Putnam, 1929. $4.50.
Gwathmey, J. T.	Textbook of anesthesia. New York, Macmillan, 1929. $7.50.
Gwinn, C. D.	Textbook of exodontia. Philadelphia, Lea & Febiger, 1927. $2.75.
Gysi, Alfred	Special teeth for cross-bite cases. New York, Dentists' Supply Co., 1928. (Pamphlet.) (No purchase orders accepted.)
Hackh, I. W. D.	Chemical reactions and their equations. Philadelphia, Blakiston, 1928. $2.00.
Haden, R. L.	Dental infection and systemic disease. Philadelphia, Lea & Febiger, 1928. $2.50.
Hanau, R. L.	Dental engineering. Buffalo, N. Y., Author, 1926. (No purchase orders accepted.)
Hanau, R. L.	Hanau intraoral method vs. Gysi extraoral method. Buffalo, N. Y., Author, 1927. (No purchase orders accepted.)
Hartman, L. L.	Teeth and the mouth. New York, Appleton, 1927. $1.50.
Head, Joseph	Everyday mouth hygiene. Philadelphia, Saunders, 1925. $1.00.
Hedstrom, Gustav	Gangrene of dental pulp and its treatment. Stockholm, Sweden, Isaac Marcus, 1927. (No purchase orders accepted.)
Headridge, Davis, & Gibson, S. K.	Dental anatomy, human and comparative. Outlines of dental science. Vol. 6. New York, Wood, 1928. $2.50.
Herbst, Emil, & Apffelsteadt, Max	Atlas and grundriss der missbildungen der kiefer and zähne. München, J. F. Lehmanns, 1928. (No purchase orders accepted.)
Hertzler, E.	Technic of local anesthesia. St. Louis, Mosby, 1925. $5.50.

Hess, Walter, & Zurcher, Ernst	Anatomy of the root-canals of the teeth. New York, Wood, 1925. $5.50.
Hewer, C. L.	Recent advances in anaesthesia and analgesia. Philadelphia, Blakiston, 1932. $3.50.
Hildebrand, G. Y.	Studies in masticatory movements of human lower jaw. Berlin, Gruyter, 1931. (No purchase orders accepted.)
Hodgen, J. D.	Practical dental metallurgy. St. Louis, Mosby, 1932. $3.75.
Hogeboom, F. E.	Practical pedodontia or juvenile operative dentistry and public health dentistry. St. Louis, Mosby, 1927. $3.50.
Hyatt, T. P.	Hygiene of mouth and teeth. Brooklyn, N. Y., Brooklyn Dental Pub. Co., 1929. $1.00.
Hyatt, T. P.	Prophylactic odontotomy: A practical and simple procedure for the prevention of caries in pit and fissure cavities. New York, Macmillan, 1933. $1.50.
Ivy, R. H., & Curtis, Lawrence	Fractures of the jaws. Philadelphia, Lea & Febiger, 1931. $4.50.
Jamieson, J. D. H.	Operative dentistry. Outlines of dental science. Vol. 9. New York, Wood, 1927. $2.50.
Jerman, E. C.	Modern x-ray technic. St. Paul, Bruce Pub. Co., 1928. $5.50.
Jordan, E. O.	Textbook of general bacteriology. Philadelphia, Saunders, 1931. $6.00.
Jordan, M. Evangeline	Operative dentistry for children. Brooklyn, Dental Items of Interest Pub. Co., 1927. $5.00.
Kanner, Leo	Folklore of the teeth. New York, Macmillan, 1928. $4.00.
Kells, C. E.	Dentists' own book, including a complete bookkeeping and recording system. St. Louis, Mosby, 1925. $7.50.
Kells, C. E.	Three score years and nine. New Orleans, The Author, 1926. $8.00.
Kennedy, Edward	Partial denture construction. Brooklyn, N. Y., Dental Items of Interest Pub. Co., 1928. $7.50.
Kent, E. N.	Business side of dentistry. St. Louis, Mosby, 1929. $5.00.
Korkhaus, Gustav	Moderne orthodontische therapie. Berlin, Meusser, 1932.
Kronfeld, Rudolf	Histopathology of the teeth and their surrounding structures. Philadelphia, Lea & Febiger, 1933. $7.00.
Kuttler, Yury	Mis investigaciones sobre la denticion en 15,240 ninos y 782 adultos habiendo hecho 28,557 examenes. Mexico, D. F., Facultad Odontologica de la Universidad Nacional Autonoma de Mexico, 1933.
LeGro, A. L.	Ceramics in dentistry. Brooklyn, N. Y., Dental Items of Interest Pub. Co., 1926. $7.50.
Leriche, R., & Policard, A.	Normal and pathological physiology of bone. St. Louis, Mosby, 1928. $5.00.
Leuck, Miriam S.	Study of dental clinics in the United States: 1930. Chicago, University of Chicago Press, 1932. $1.00.
Leuck, Miriam S.	Further study of dental clinics in the United States. Chicago, University of Chicago Press, 1932. $1.50.
Leven, Maurice, & Beck, Dorothy F.	Practice of dentistry and incomes of dentists in twenty states: 1929. Chicago, University of Chicago Press, 1932. $2.00.
Lindsay, Lilian	Short history of dentistry. London, John Bale, 1933. $1.50.
Livingston, Alexander	Dental histology. Outlines of dental science. Vol. 10. New York, Wood, 1928. $2.50.
Lloyd-Williams, E.	Dental mechanics: a manual for students and junior practitioners. London, Bale, 1931. $2.00.
Logan, J. D.	Dental prosthetics. Outline of dental science. Vol. 4. Edinburgh, Livingstone, 1926. $2.50.
Lucas, H. A.	Medicine for dental students. Baltimore, William Wood & Co., 1933. $2.75.
Margolis, H. M.	Conquering arthritis. New York, Macmillan, 1932. $2.00.

Marriott, W. M.	Infant nutrition: a textbook of infant feeding for students and practitioners of medicine. St. Louis, Mosby, 1930. $5.50.
Marsden, P. H.	Dental materia medica. Outlines of dental science. Vol. 3. Edinburgh, Livingstone, 1926. $2.50.
Marshall, J. A.	Diseases of the teeth; their diagnosis and treatment. Philadelphia, Lea & Febiger, 1926. $4.50.
Maximow, A. A., & Bloom, Wm.	Textbook of histology. Philadelphia, Saunders, 1930. $9.00.
McBride, W. C.	Juvenile dentistry. Philadelphia, Lea & Febiger, 1932. $2.75.
McCaw, Emma J.	Dental assistant. St. Louis, Mosby, 1926. $1.50.
McCollum, E. V., & Simmonds, Nina	Food, nutrition and health. Baltimore, Authors, 1928. $1.50.
McCollum, E. V., & Simmonds, Nina	Newer knowledge of nutrition. New York, Macmillan, 1929. $5.00.
McCoy, J. D.	Applied orthodontia. Philadelphia, Lea & Febiger, 1931. $4.50.
McGehee, W. H. O.	Textbook of operative dentistry. Philadelphia, Blakiston. 1930. $10.
Mead, S. V.	Diseases of the mouth. St. Louis, Mosby, 1932. $10.00.
Mellanby, May	Diet and the teeth: an experimental study. Part I. Dental structure in dogs. London, His Majesty's Stationery Office, 1929. $7.00.
Mellanby, May	Diet and the teeth: an experimental study. Part II. A. Diet and dental disease. B. Diet and dental structure in mammals other than the dog. London, His Majesty's Stationery Office, 1930. $1.00.
Merritt, A. H.	Periodontal diseases: diagnosis and treatment. New York, Macmillan, 1930. $4.00.
Moorehead, F. B., & Dewey, Kaethe W.	Pathology of the mouth. Philadelphia, Saunders, 1925. $7.00.
Neil, Ewell	Full denture practice. Nashville, Tenn., Marshall and Bruce Co., 1932.
Nevin, Mendell, & Puterbaugh, P. G.	Conduction, infiltration and general anesthesia in dentistry. Brooklyn, Dental Items of Interest Pub. Co., 1927. $6.00.
Nichols, I. G.	Prosthetic dentistry: an encyclopedia of full and partial denture prosthesis. St. Louis, Mosby, 1930. $12.50.
Okumura, Tsurukichi	Present status of dentistry in Japan. Tokyo, Japan, Japan Dental Assn., 1931. (Pamphlet.) (No purchase orders accepted.)
Orban, Balint	Dental histology and embryology. Philadelphia, Blakiston, 1929. $5.00.
Osgood, H. A.	Teeth and jaws roentgenologically considered. Annals of roentgenology, volume 5. New York, Hoeber, 1929. $10.00.
Ottolengui, Rodrigues	Table talks on dentistry. Brooklyn, Dental Items of Interest Pub. Co., 1928. $7.00.
Palmer, G. T., Derryberry, Mahew & Van Ingen, Philip	Health protection for preschool child: national survey of use of preventive medical and dental service for children under six. New York, Century, 1931. $2.50.
Perry, Maude A.	Dietetics and nutrition. St. Louis, Mosby, 1930. $2.50.
Phelps, Edith M.	Socialization of medicine. New York, Wilson, 1930. $0.90.
Pollia, J. A.	Fundamental principles of alveolo-dental radiology. Brooklyn, Dental Items of Interest Pub. Co., 1930. $8.00.
Posner, J. J.	Minor oral surgery simplified. Philadelphia, Patterson & White, 1928. $2.00.
Prinz, Hermann	Dental formulary. Philadelphia, Lea & Febiger, 1930. $3.50.
Prinz, Hermann	Dental materia medica and therapeutics. St. Louis, Mosby, 1926. $7.50.
Prinz, Hermann	Diseases of soft structures of teeth. Philadelphia, Lea & Febiger, 1928. $6.50.
Prothero, J. H.	Prosthetic dentistry. Chicago, Medico-Dental Pub. Co., 1928. $10.00.
Pusey, W. A.	Doctor of the 1870's and 1880's. Baltimore, C. C. Thomas, 1932. $3.00.
Quigley, D. T.	Notes on vitamins and diets. Chicago, Consolidated Book Publishers, Inc., 1933. $1.00.
Raper, H. R.	Clinical preventive dentistry. Rochester, N. Y., Ritter Dental Mfg. Co., 1926. $3.50.

Rapp, F. W.	Elements of anatomical articulation or the mathematics of dental prosthetics. London, Bale, 1930. $2.00.
Ray, K. W.	Metallurgy for dental students. Philadelphia, Blakiston, 1931. $3.00.
Report of Committee on Growth and Development	Growth and development of the child. Part III. Nutrition. (A publication of White House Conference on Child Health and Protection.) New York, Century, 1932. $4.00.
Report of Committee on Public Health Organization	Public health organization. (A publication of White House Conference on Child Health and Protection.) New York, Century, 1932. $3.00.
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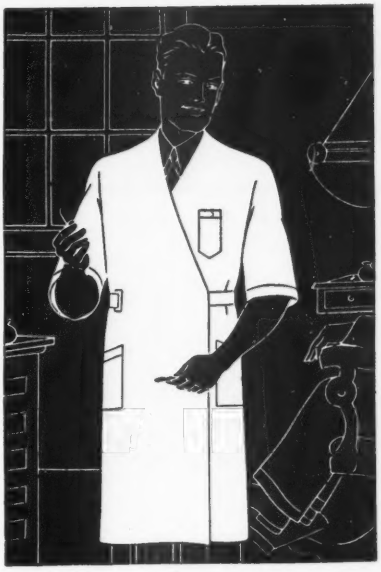

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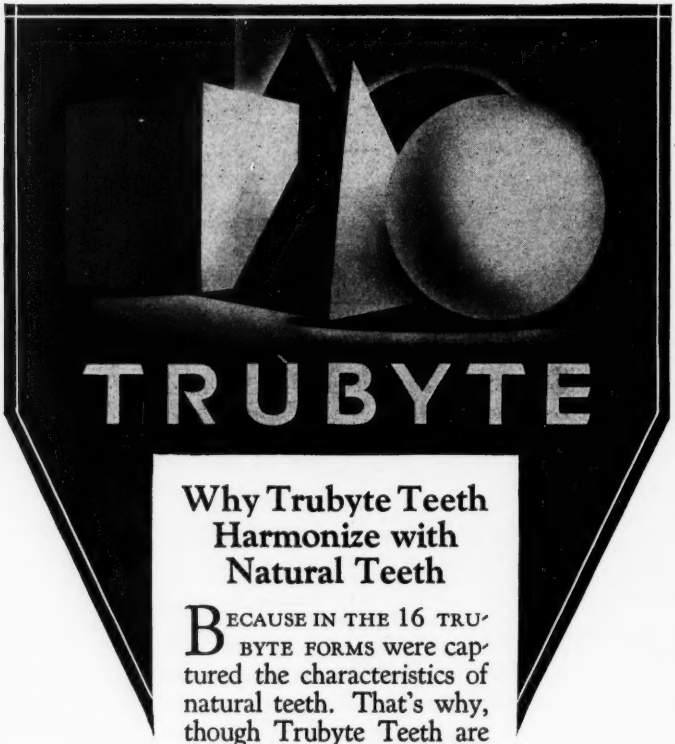

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